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JAMES PETER JOLIVETTE

1915–1945

J. C. WALKER

James Peter Jolivette was born near LaCrosse, Wisconsin, on July 20, 1915, the son of Bert A. and Vanetta Jolivette. After completing his secondary school training at LaCrosse High School in 1933, he entered the University of Wisconsin in the autumn of that year. Early recognition

JAMES PETER JOLIVETTE
1915–1945

of his ability came in the form of the Steenbock Scholarship Award, which is granted annually to a Senior in the College of Agriculture outstanding in scholarship, leadership, and promise. He received the Bachelor of Science degree in Agriculture, with a major in Soils in June, 1937. Enter-

ing the Graduate School in the same institution in September, 1937, he completed his Doctorate, with a major in Plant Pathology and a minor in Botany, in June, 1941.

In the spring of 1941, Jolivette assumed immediate charge of the truck crop disease field laboratory in southeastern Wisconsin, left vacant by the death of Dr. Otis C. Whipple on February 13 of that year. He was appointed Instructor in Plant Pathology in July, 1941.

Having been a member of the Reserve Officers' Training Corps, as an undergraduate, Jolivette held a commission as Second Lieutenant in the Officers' Reserve Corps, U. S. Army, from the time of his graduation in 1937. Continuing his training in ORC during the period of his graduate studies, he received a promotion to First Lieutenant in July, 1941. Early in 1942 he was called into service and reported for active duty on March 20, 1942. After three months in the Officers' Training School at Fort Benning, Georgia, he was assigned to Company L, 20th Infantry, 6th Division, U. S. Army. In May, 1943, he became Captain of that Company and continued in that capacity until his death. After about a year of intensive training in this country his Division left the Continental United States in September, 1943. Following a brief stay in the Hawaiian Islands, his Company entered the New Guinea campaign early in 1944. During this campaign he was awarded the Silver Star. On January 9, 1945, American troops invaded Luzon, P. I., at Lingayen Gulf and Company L participated in this landing. On February 2, 1945, Captain Jolivette was killed by enemy fire at Munoz, P. I., while conducting a volunteer detachment in the rescue of wounded men and officers. Burial was in the Santa Barbara cemetery near by. He was awarded posthumously the Purple Heart Decoration and a second Silver Star citation.

During his scientific career, Jolivette was engaged in research on vegetable diseases. He carried out very creditable work in the boron deficiency diseases of beet, cabbage, and other vegetables. He studied critically the histological changes brought about in growing plants which were deficient in boron and took an active part in disease resistance breeding programs under way with cabbage, tomato, and bean.

As an investigator he showed marked ability and originality. He took the lead among his associates in the application of modern statistical methods to the design of pathological experiments and to the analysis of results. He showed unusual strength in making contacts with growers and vegetable canners. His field of science has been deprived of a recruit who had won his spurs as an investigator and showed great promise both in fundamental research and in its applications to the agricultural industry.

Captain Jolivette was married on August 23, 1939, to Eloise Lauson. He is survived by his wife and by two sons, Peter Lauson, born May 27, 1941, and David James, born December 11, 1942.

A man of kindly and reserved temperament, his loss is felt keenly by his many scientific colleagues and his acquaintances in the agricultural

industry. Plant Pathology has lost a promising recruit in a man who held defense of his country as his first duty. His record remains as an inspiration, alike to those who knew him and to younger men who follow him into this field.

Research papers in which Jolivette was an author are as follows:

Internal black spot of canning beets and its control. Canning Age **19**: 489–491; 508. 1938. (With J. C. WALKER and J. G. MCLEAN.)

Studies on boron deficiency in garden beet (*Beta vulgaris*). (Abstr.) Phytopath. **31**: 23. 1941. (With J. C. WALKER.)

The boron deficiency disease in cabbage. Jour. Agr. Res. [U.S.] **62**: 573–587. 1941. (With J. C. WALKER and JOHN G. MCLEAN.)

Boron deficiency in garden and sugar beet. Jour. Agr. Res. [U.S.] **66**: 97–123. 1943. (With J. C. WALKER and JOHN G. MCLEAN.)

Effect of boron deficiency on the histology of garden beet and cabbage. Jour. Agr. Res. [U.S.] **66**: 167–182. 1943. (With J. C. WALKER.)

Productivity of mosaic-resistant Refugee beans. Phytopath. **33**: 778–788. 1943. (With J. C. WALKER.)

The limitations of spraying tomatoes in Wisconsin. Wis. Agr. Exp. Stat. Res. Bull. 152. 1944. (With J. C. WALKER, O. C. WHIPPLE, and W. J. HOOKER.)

Varietal susceptibility in garden beet to boron deficiency. Soil Science **59**: 461–464. 1945. (With J. C. WALKER and W. W. HARE.)

Yellows-resistant cabbage varieties in the early and mid-season round head groups. U. S. Dept. Agr. Tech. Bull. (In press.)

SOME FEATURES OF THE SPREAD OF PLANT DISEASES BY AIR-BORNE AND INSECT-BORNE INOCULUM

E. E. Wilson and G. A. Baker[1]

(Accepted for publication January 21, 1946)

Visual evidence of the spread of phytopathogenic fungi by wind is afforded in the development of the diseases on above-ground parts of susceptible plants near a source of inoculum. Plant pathologists have frequently reported the disease zones or gradients that appear in such location, but few have given the phenomena critical attention. They agree, however, that the decrease in the incidence of infection as distance from the source of inoculum increases is probably associated with a corresponding decrease in the density or frequency of the inoculum in the air during the dissemination period or periods. If this is true, it follows that the characteristics of the disease gradients might reflect the pattern of spore dispersion,[2] but since the disease results from the interplay of a large number of factors, some affecting spore dissemination and others influencing infection and the development of symptoms, the infection pattern would probably not possess features identical in all respects with those of the aerial spore pattern. According to Gregory (8), however, certain features of the gradients reported for various diseases are noticeably constant. This he believes is due in large measure to a fairly constant pattern of spore dissemination. Gregory (8), moreover, analyzes some of the published results on different disease and aerial spore density gradients, and shows a correspondence between these data and features pertaining to the transfer of atmospheric properties (heat, momentum, and suspended particles) by eddy diffusion.

The present writers (19) attempted to show the relationship between the spatial distribution of blossom blight, caused by *Sclerotinia laxa* Ader. and Ruh., in groups of apricot trees, and the pattern of spore dispersion; paying particular attention to effects of wind velocity on the pattern of spore dispersion. When a study of data collected by others was undertaken, however, it became apparent that the individual effects of the numerous factors involved could not be determined, because information regarding these factors was not given in the published reports. It was desirable, therefore, for the analyses of such data to follow a procedure that would provide for the effects of a large number of undetermined variables without being cumbersome.

The writers wish to describe a method based on the results of their (19) earlier studies of spore dispersion and to compare this method with that

[1] Associate Plant Pathologist and Assistant Statistician, respectively.

[2] The scattering of spores in the dimensions transverse to the main direction of the wind. Meteorologists employ the term "diffusion" to describe a similar process in the transfer of atmospheric properties by eddy movement.

described by Gregory (8). Inasmuch as the mathematical procedures involved are more or less standard, they are not described in detail.

CORRELATING THE PATTERN OF DISEASE INFECTION WITH THE PATTERN OF SPORE DISSEMINATION

Seven gradients of brown-rot blossom blight were reported by the writers (19); 3 for 1939 and 4 for 1940. Though the rate of decrease of infection with distance was greater in 1939 than in 1940, the curves representing the incidence of blossom infection in trees receiving the inoculum, when expressed as percentages of the incidence in source trees, were described by the equation:

$$y = \frac{A}{x^p} \tag{1}$$

where y was the incidence in recipient trees, x the distance in feet from the center of the nearest source trees, and A and p were constants depending on the wind velocity during the period of "effective dissemination,"[3] and perhaps on other quantities to a lesser extent.

According to results obtained by releasing spores and intercepting them at different distances downwind, the aerial density,[4] y, of these bodies diminished about inversely proportional to the square of the distance from the source. Hence, if the density at the station, x, nearest the source at which spores were intercepted was considered to be 100, the densities at subsequent distances, expressed as percentages of the density at x_1, were described by

$$y = \frac{100}{x^2}. \tag{2}$$

According to other tests (19), moreover, the dispersion of air-borne spores, over open ground at least, increased approximately proportional to the distance from the source. Thus, the chances for infection of recipient plants downwind from the source of spores would probably diminish inversely proportional to some power of x, the distance from the source.

In the studies (19) of apricot blossom blight it was possible to determine the incidence of infection at the source. With many diseases, however, this is frequently impossible, since the source may be an alternate host of the fungus, a group of plants differing in susceptibility from the recipient plants, or in some instances refuse such as piles of potato tubers (2). In order to compare the rate at which infection diminishes with distance, therefore, it is necessary to take as a reference point the incidence of infection at the location nearest the source at which observations are made. This was the procedure adopted for studying aerial spore densities. In these spore dissemination studies the distances from the source (point of release) were $x_1 = 5$, $x_2 = 10$, and $x_3 = 15$ feet. As it happened, the density values obtained at these distances led to the relationship expressed by

[3] Dissemination resulting in the initiation of infection in the recipient hosts.

[4] The number of spores passing through unit area of a plane transverse to the mean direction of the wind.

equation 2. Suppose, however, that instead of intercepting the spores at 5, 10, and 15 feet, they were caught at 10, 20, and 30 feet. In the first case the spore density values at 10 and 15 feet would be expressed as percentages of the value obtained at 5 feet, and in the second case the values at 20 and 30 feet would be expressed as percentages of the density obtained at 10 feet. In both cases, however, the relative distances would be the same,

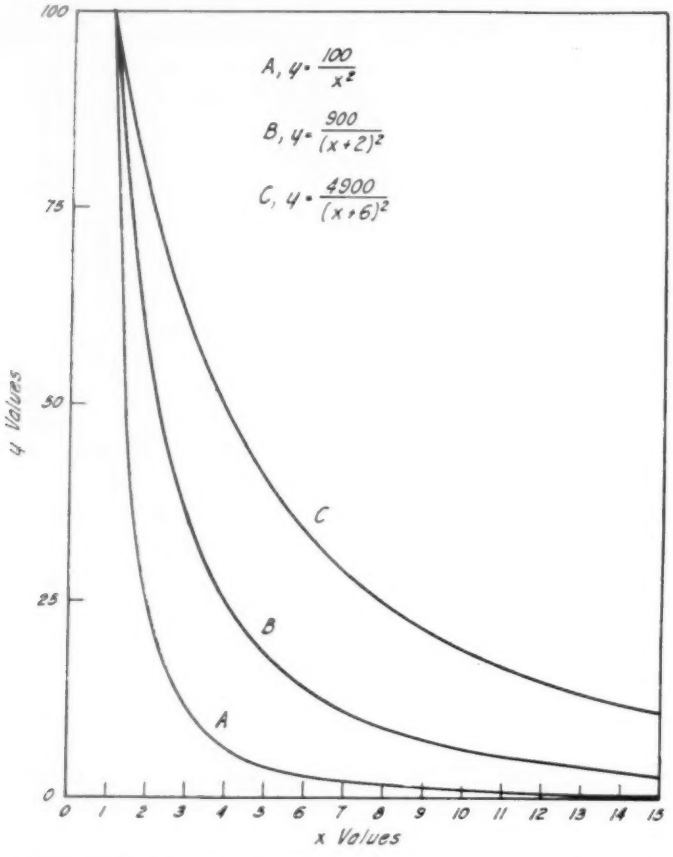


FIG. 1. Curves expressing the density, y, of air-borne spores at increasing distances, x, from the source of spores. The unit in which x is measured is the distance from the source to the nearest station, x_1, at which observations are made. The critical distance from the source is such that if x_1 is at this distance the observed data will follow A, the inverse square law. Increasing x_1 by two or six times the critical distance, on the other hand, produces curves B and C respectively.

1, 2, and 3, etc. If, as indicated by experiments (19), the density values obtained at 5, 10, 15, etc., feet from the source were expressed by equation 2, the results would plot curve A, figure 1. Judging from this curve, the rate at which the values approach the x axis diminishes rapidly as absolute distance from the origin increases. If, therefore, spore density was measured at 10, 20, and 30 feet, the slope of the gradient would be less than that represented by curve A. Since the same relative distances are

involved, the curve would originate at $x = 1$, but for successive values of x it would approach the x axis less rapidly than A. In order to adjust for the effects of varying the absolute distance from the source to the first observation station, the quantity a is introduced into equation 2 as follows:

$$y = \frac{100\ (1+a)^2}{(x+a)^2}. \qquad (3)$$

To determine the curve of best fit for results which vary with respect to the location of x, some rule for selecting a must be found. Equation 3 contains only one constant, a, to be determined, so we are to impose a single condition, the simplest and most desirable of which, perhaps, is that the sum of the differences between the observed and theoretical values be zero. The method for calculating a is a standard mathematical procedure.

To illustrate, the slopes of curves produced by equation 3, curves A, B, and C in figure 1 are plotted for a values of 0, 2, and 6 respectively. Increases in the value of a decrease the slopes of the curves.

In as much as the original data and details regarding the circumstances under which results were secured can be obtained from the literature cited, the presentation herein is confined to the percentage relationships between disease development at various relative distances from the source of inoculum. These percentages are computed from the original data, which in some cases were tabulated but in others appeared in figures and maps.

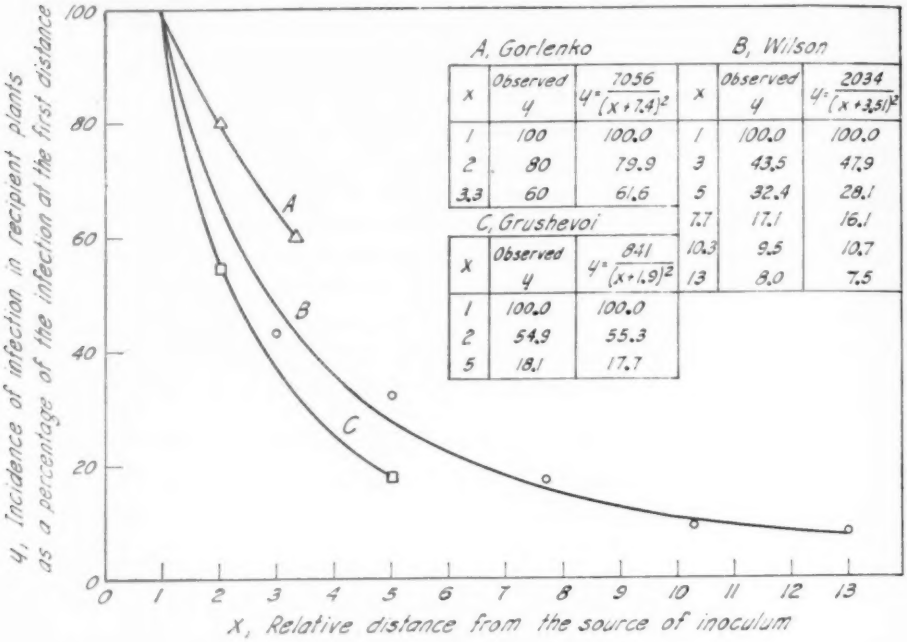
DISEASE GRADIENTS RESULTING FROM AIR-BORNE INOCULUM

According to Stepanov (17), both Gorlenko and Grushevoi determined the spread of crown rust of oats in one direction from the alternate host, *Rhamnus sp.* Grushevoi's results are expressed as the average "grade" of infection in an area "immediately neighboring" the source and at 10 and 25 "sazhen" (70 and 175 feet) from the source. Since this leaves only two points on which to establish a curve, Grushevoi's data are of little value. In figure 2, however, the curve is drawn on the assumption that the first point was 5 "sazhen" from the source. The distance at which Gorlenko made his first observations is not given, so computations are based on infection at the second distance, 45 meters.

Through the kindness of Dr. C. A. Suneson of the Division of Cereal Crops and Diseases, U. S. Department of Agriculture, the senior writer observed in the spring of 1945 a case where crown rust spread from groups of wild oat plants growing along an irrigation ditch running east and west. These plants, having obtained moisture from the ditch in late summer and early autumn, were much earlier to develop than other oats in the vicinity. They were infected by *Puccinia coronata*, probably in the autumn of 1944 before oats in the vicinity started development. In any event, a marked gradient of new infection by the crown rust fungus extended from these plants northward through the stand of less mature plants. In April the senior writer secured one set of counts on the number of lesions per 100 sq. cm. of leaf surface in recipient plants at different distances from the

edge of a group of source plants extending for a distance of 3 feet along the ditch bank. Unfortunately, further counts were precluded because lesions resulting from secondary infection appeared shortly thereafter. The results, together with the curve of best fit, appear in figure 2.

Although the number of points at which the Russian workers made their observations are few, and the first point in Grushevoi's data is an assumed value, curves are fitted to them very satisfactorily by equation 3. The senior writer's results also are fitted satisfactorily. Here the number of



A, Gorlenko			B, Wilson		
x	Observed y	$y=\frac{7056}{(x+7.4)^2}$	x	Observed y	$y=\frac{2034}{(x+3.5)^2}$
1	100	100.0	1	100.0	100.0
2	80	79.9	3	43.5	47.9
3.3	60	61.6	5	32.4	28.1
			7.7	17.1	16.1
C, Grushevoi			10.3	9.5	10.7
x	Observed y	$y=\frac{841}{(x+4.9)^2}$	13	8.0	7.5
1	100.0	100.0			
2	54.9	55.3			
5	18.1	17.7			

FIG. 2. Gradients of crown rust infection on oat plants in the vicinity of buckthorn bushes (Gorlenko and Grushevoi) and on young wild oats in the vicinity of more mature wild oat plants which served as a source of spores (Wilson).

observation points were more numerous and, consequently, the trend of the data appears in greater detail.

Results reported by Buchanan and Kimmey (3) and Posey and Ford (15) pertaining to the spread of the blister rust from currant bushes to pine trees are represented fairly well by equation 3 (Fig. 3), the major difference between observed and computed results being in the tendency for the slope of the gradient at two or three points near its outer boundary to be greater than that of the curve. For example, in curve A from relative distances 1 to 11 the trend of the observed percentages is reasonably well represented by the curve, but for relative distances 13 to 17 the observed values decrease more rapidly than the curve. Similar tendencies were noticeable in the gradient north and south of the source, and in gradients of a few other diseases. Though one might conclude that such

data might be more exactly represented by an equation in which the exponent of x was greater than 2, the rapid decrease in the value obtained at the outer boundary of a gradient is possibly due to chance events such as the nonuniform distribution or grouping of the recipient hosts, or to observations on insufficient numbers of individuals.

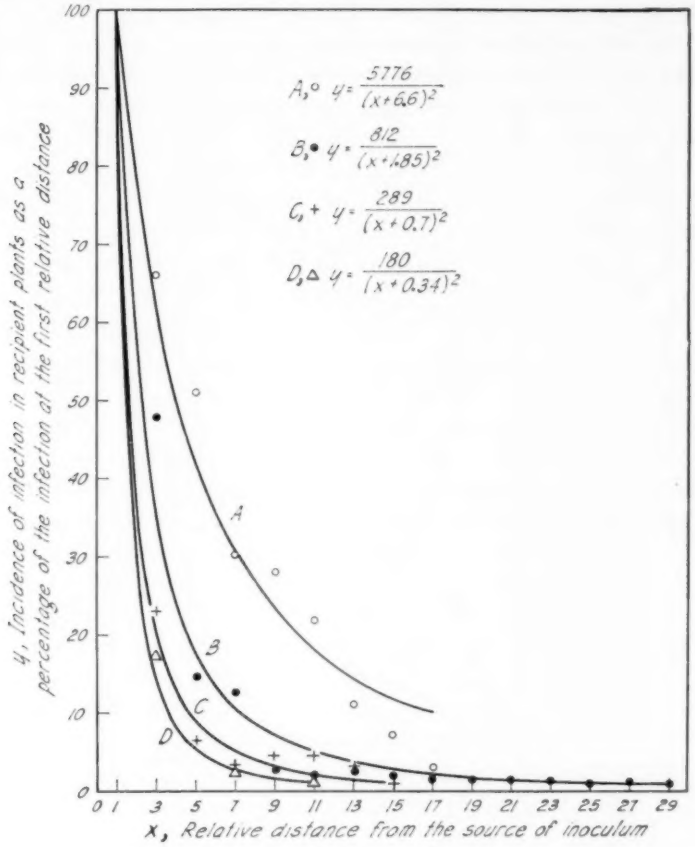

A, ○ $y = \frac{5776}{(x+6.6)^2}$
B, ● $y = \frac{812}{(x+4.85)^2}$
C, + $y = \frac{289}{(x+0.7)^2}$
D, △ $y = \frac{180}{(x+0.34)^2}$
y, Incidence of infection in recipient plants as a percentage of the infection at the first relative distance
x, Relative distance from the source of inoculum
A
B
C
D
0 1 3 5 7 9 11 13 15 17 19 21 23 25 27 29
0 10 20 30 40 50 60 70 80 90 100

FIG. 3. A and C, gradients of blister rust infection in pine trees, west and east, respectively, of a group of currant bushes (Posey and Ford). B, a gradient of blister rust infection in pine trees surrounding a group of currant bushes (Buchanan and Kimmey). D, a gradient of late blight infection in a potato field near a pile of refuse potatoes.

Bonde and Schultz (2) observed the spread of late blight, caused by *Phytophthora infestans* from refuse piles of potato tubers to nearby potato fields. Table 3 of their paper reports the percentage of recipient plants infected at various distances from the source and the number of lesions per 100 plants at these distances for one field. While the percentage of plants infected cannot be represented by equation 3, the data on the number of lesions is roughly fitted. On the other hand, the average percentages of plants infected in five other fields are well represented by equation 3, as can be seen in curve D, figure 3.

The foregoing data were selected as fair representations of the apparent general agreement between observed results and the concept that a basic pattern is reflected in most of the disease gradients. Further indications of this are seen in Boevsky's (1) studies of the spread of leaf rust of wheat (results for June 13). Here the percentage infection at 50, 100, and 200 meters from the source corresponds very closely to equation 3 with an a value of 4. Results on the spread of tulip spot given by Wallace (18) and compiled by Gregory (8, table 13, observations for April 4) are in good

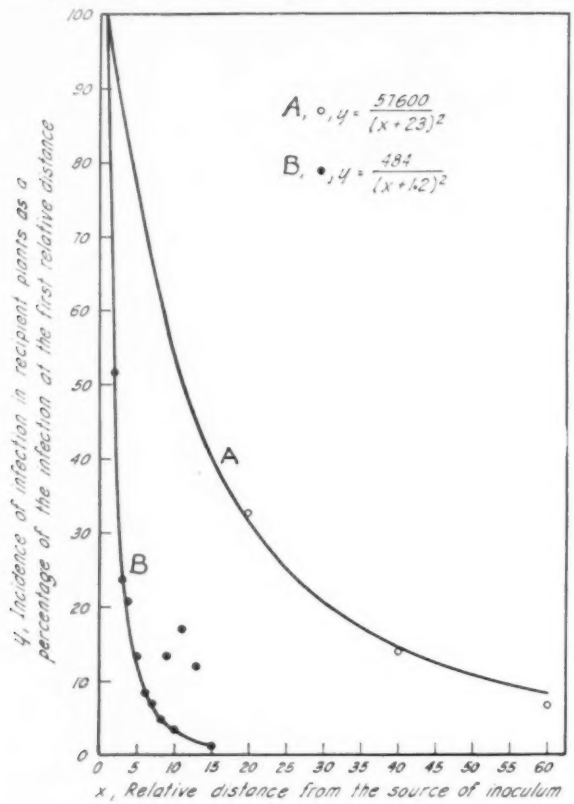


FIG. 4. A, a gradient of yellow dwarf infection in a potato field adjacent to a meadow from which the insect vector migrated (Frampton, Linn, and Hansing). B, a gradient of angular leaf spot infection in cotton plants east of a row inoculated with the causal bacterium (Faulwetter).

agreement with equation 3, having an a value of 0.76. Two out of three gradients of loose smut of wheat reported by Oort (13) are described fairly well by equation 3 with a values of 2.84 and 6.5.

In certain cases reported by other workers the percentage infection at successive distances decreases irregularly, and in consequence, no type of smooth curve can be made to pass through more than a few of the observed values. By and large, however, the general trend of the data is expressed by equation 3 in the following cases: cedar-apple rust (16), downy mildew

of onion (11), powdery mildew of barley (14), and eye spot of wheat (12). On the other hand, the spread of black stem rust of wheat from barberry to wheat reported by Johnson and Dickson (9) is not represented by equation 3.

According to Faulwetter (4), the bacterium, *Phytomonas malvacearum,* causing angular leaf spot of cotton is spread in wind-blown droplets of rainwater spattered from the surface of infected leaves. Faulwetter (5) presents maps illustrating the spread of this disease from a row of inoculated plants in the middle of a cotton field. From Faulwetter's figure 1 the present writers compiled data pertaining to the number of lesions per plant in 14 successive rows east of the portion of the row inoculated with cultures 140, 143, 148, and 151, and in 4 successive rows east of the portion inoculated with cultures 141, 146, 149, and 153. Heavy infection of a few plants in rows 9, 11, 12, and 13 raised the averages for these rows. The location of these plants in groups suggested the possibility of natural infection having occurred on certain plants in these areas and a subsequent spread to surrounding plants. When the high values in these four rows were included in the calculations of the a value, the resulting curve followed the trend of the majority of points but passed above the lower values. Omitting the high values from computations, however, resulted in a satisfactory fit (lower curve, figure 4), the a value being 1.2. Data from the four rows east of cultures 141, 146, 149, and 153 were described very closely by an equation in which $a = 2.01$.

GRADIENTS FOR DISEASES SPREAD BY INSECT-BORNE INOCULUM

At first thought the spread of plant disease by means of an insect vector would appear to have little in common with the spread of disease by air-borne spores. The individual insect flight or hop would seem to be so erratic as to preclude a sustained spread in any one direction. In cases where the insects develop on one crop and, obtaining the pathogen on this crop, migrate to another, however, there is an expansion, in some cases unilaterally, of the area in which the insect occurs. According to studies by Linn (10) and Frampton, Linn, and Hansing (6), the migration of viruliferous leafhoppers from source or reservoir plants into adjacent plantings of susceptible crops, results in distinct gradients of infection. Linn (10) noted that the percentage of yellows in successive rows of endive plants in the vicinity of weeds from which leafhoppers had migrated "was in inverse ratio" to the distance of the row of endive from the weeds. Frampton, Linn, and Hansing (6) compare the spread of these insects across the boundary between two crop areas to the flow of a compressible fluid and the components of this spread—the average length of insect hop—to the mean free path of gas molecules. It might be said here that such a concept is similar to that regarding the movement of atmospheric "turbulence bodies" or eddies which are responsible for the dispersion of air-borne objects (7). In developing an equation to express the spread of virus

diseases of the yellows type, Frampton, Linn, and Hansing (6) employ the concept of an unilateral diffusion process.

It is not the purpose to examine their treatment here, but by means of equation 3 to compare the gradients of diseases arising from the insect-spread of the inoculum with those arising from the aerial spread of the inoculum.

In his figure 9 Linn (10) plotted the percentage of yellows in rows of endive plants at increasing distances from a weedy strip from which the leafhoppers migrated. The results given in his curves A, C, and D were converted into the form suitable for application of equation 3. The trend of the results in curve B was too uncertain for a profitable treatment.

The results from curves A and D follow equation 3 to a noticeable degree, the values of a differing but little. In curve C, however, the data do not follow this relationship but seemingly are best described by an equation for a straight line.

Frampton, Linn, and Hansing (6) illustrate (figure 1 of their paper) the manner in which yellow dwarf was spread from a meadow into a potato field by the clover leafhopper. For the first 60 potato rows the percentage of plants infected progressively decreases as distance from the meadow increased, but because of the entry of leafhoppers from an uncultivated strip on the side of the potato field opposite the meadow, the incidence of disease increased somewhat in subsequent rows. The data to be examined, therefore, are those of rows 1, 20, 40, and 60.

The exact distance of row 1 from the edge of the meadow is not given, although the width of the rows is said to be 18 inches. Consequently, one cannot compare infection as to relative distances as we have been doing. It is possible, however, to fit the data in relation to the incidence of disease in row 1, though a very high value for a results. When this is done, however, (Fig. 4), we see that equation 3 apparently describes the data satisfactorily.

Zentmyer, Wallace, and Horsfall (20) established plantings of young elm trees around isolated old trees which were infected by the Dutch elm disease and also were infested by the insect vector of this disease, *Scolytus multistriatus*. Two years later they determined the percentage of young trees attacked by the disease in concentric zones whose outer boundaries were respectively 25, 75, 175, and 320 feet from the source trees. The mean percentages of 3 such plots were taken from their table 1. There is only fair agreement between observed and calculated results because the percentage at the last relative distance drops very rapidly, an irregularity noted in other disease gradients.

COMPARISON OF EQUATION 3 WITH AN EQUATION FOR THE TRANSFER OF ATMOSPHERIC PROPERTIES BY EDDY DIFFUSION

The transfer of heat, momentum, and matter by eddy diffusion in relation to the dispersion of spores is discussed in a recent paper by Gregory

(8). A correspondence is shown between the pattern of spore dispersion and the characteristics of disease gradients on the one hand and the characteristics of eddy diffusion on the other hand. It is proposed to compare Gregory's treatment with that employed in the present paper. Gregory begins by assuming the effect of gravity on most air-borne spores to be small in comparison to the effects of forces which keep them afloat.[5] Thus, clouds of air-borne spores may be regarded as suspensions in the atmosphere. Consequently, their dispersion from a source should follow a pattern similar to that followed in the dispersion of smoke by eddy diffusion. For a more detailed explanation of this procedure reference should be made to Gregory's (8) paper. Its essential features are as follows: Consider the emission of air-borne particles from a point at the origin of x, y, z coordinates (x being horizontal to the ground in the mean direction of the wind, y being horizontal to the ground and at right angles to the mean direction of the wind, and z being vertical to the ground). A quantity of spores is liberated as a large number of small clouds, which may or may not take the same direction, but which diffuse as they travel downwind. The standard deviation of the positions of the spores from their mean position (the x axis) at any given distance from the source is given by $\sigma^2 = \frac{1}{2}C^2x^m$. According to Gregory (8), the gradient of spore deposition at all distances, x, from the point source will be described by:

$$d_w = \frac{p\,2\,Q}{\pi C^2 x^m}$$

where

d_w = mean deposition per sq. cm. straight downwind at distance x from the source.

p = a deposition coefficient or the proportion of spores that will be deposited out of the total number crossing 1 sq. cm. of surface.

Q = the total number of spores liberated.

C = a coefficient of diffusion regarded as constant by Gregory.

m = a number between 1 and 2 regarded as constant for any set of conditions and any particular time interval.

This equation describes diffusion from a point source. For diffusion from a line source at right angles to the wind, the treatment must be modified. However, the equation for a point source will apply if the distance from the source is more than four times the dimension of the line source (8, p. 42). Gradients for strip and block sources will be flatter than those for point or line sources. Such differences in the steepness of the gradient are determined by the exponent of x.

The quantity m requires some discussion. On page 41 Gregory says that m is an indicator of the degree of atmospheric turbulence and to a first approximation is independent of wind velocity. It is primarily affected only by those factors that tend to dampen or enhance turbulence. It has a value

[5] Gregory presents evidence that the size of the spore, hence its rate of fall in still air, influences very little, if any, the proportion of spores reaching the ground.

of 1.24 for low turbulence and approaches 2 for extreme turbulence, a normal value being 1.75. Moreover, m increases with the length of the sampling periods. In case of dissemination periods extending over hours or days, therefore, the value of m is probably a maximum.

The quantity C is a coefficient of diffusion which is said to remain relatively constant for the layer of air near the ground. It is said to have a normal value of about 0.6.

According to the evidence in the foregoing section, equation 3 of the present paper expresses a number of disease gradients satisfactorily, but the question arises as to whether the eddy diffusion equation represents the situation more accurately. We shall first examine the results given by

$$d_w = \frac{p\,2\,Q}{\pi C^2 x^{1.75}} = \frac{p\,2\,Q}{1.13x^{1.75}} \text{ and } y = \frac{100(1+a)^2}{(x+a)^2}$$

on comparable bases. To do this we assume that for the eddy diffusion equation the relative number of spores, $p\,2\,Q$, has a numerical value of 113, thus making $d_w = 100$ when $x = 1$. The equation becomes $d_w = \frac{113}{1.13x^{1.75}}$. Curve A in figure 5 represents the d_w values of this equation for thirteen relative distances from the source, whereas curve B is the plotted values of $y = \frac{146}{(x+0.21)^2}$. The close correspondence of these curves is seen by inspection. The maximum difference between the ordinates of curves A and B occurs at $x = 7.76$, and is only 0.47 per cent.

We next consider the decrease in the slope of the eddy diffusion curve occurring when m diminishes to 1.24, during short sampling periods or when atmospheric turbulence is low. Curve C in figure 5 represents such a curve. Curve D gives the values of y for equation 3, in which $a = 1$. It is seen that the two curves correspond very closely near the source but diverge at distances farther from the source.

It is unlikely that such small differences as indicated between curves A and B will be revealed in disease infection data, or, for that matter, in data from studies of aerial density of spores. The slope of the eddy diffusion curve is slightly less than the slope of equation 3.

In general, the two procedures give very similar results so long as the value of m is 1.75 or higher. Flatter gradients are said to occur (10) when the source is a strip or a block. An examination of results given by Boevsky (1) and by Pape and Rademacher (14), regarding changes in disease gradients over a period of time, suggests that the steepness may be modified by other causes, one being secondary infection from spores produced on the recipient plants themselves. Secondary infection of course introduces complications which preclude adequate treatment by either of the two procedures.

No doubt many other factors modify the steepness of disease gradients. It should be noted that the constant a was originally introduced into equation 3 to adjust for variations in the distance of x_1 from the source. Appar-

ently, however, little or no correlation exists between this distance and the value of a. Presumably, therefore, the constant a depends upon other factors as well. According to the comparisons in the foregoing section, equation 3 appears to fit the trend of many observed data reasonably well. While the eddy diffusion equation may express the effects of meteorological factors somewhat more closely than equation 3, it does not cover the great differences in slopes exhibited by the observed results. For example, the

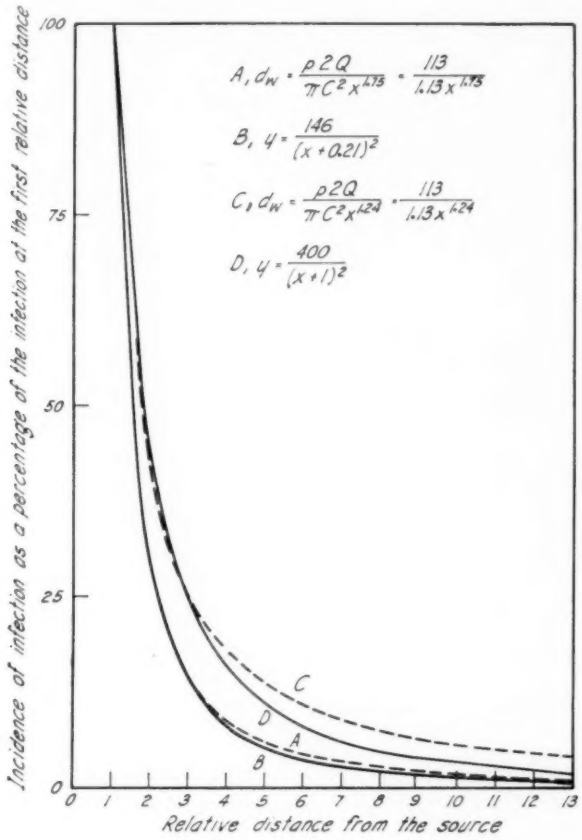

Incidence of infection as a percentage of the infection at the first relative distance
100
75
50
25
0
0 1 2 3 4 5 6 7 8 9 10 11 12 13
Relative distance from the source
A, $d_w = \frac{p2Q}{\pi C^2 x^{1.73}} = \frac{113}{1.13 x^{1.73}}$
B, $y = \frac{146}{(x+0.21)^2}$
C, $d_w = \frac{p2Q}{\pi C^2 x^{1.24}} = \frac{113}{1.13 x^{1.24}}$
D, $y = \frac{400}{(x+1)^2}$
C
D
A
B

FIG. 5. A comparison of curves produced by the modified inverse-square equation with curves produced by Gregory's eddy diffusion equation having different values for the exponent of x.

minimum slope produced by the equation for a point source is illustrated by curve C, figure 5. On the other hand, many of the disease gradient curves are much flatter than curve C. The eddy diffusion equation, modified to represent dispersion from a block source, is scarcely applicable to certain of these gradients, since the sources cannot be considered blocks according to Gregory's (8) definition.

Hence, apparently the major difficulty in applying the eddy diffusion equation arises from its failure to account for the combined effects of the

large number of factors which affect characteristics of the gradients. The family of curves produced by equation 3, on the other hand, furnishes a much more flexible tool for at least a preliminary study of disease gradients. If and when the effects of particular factors are determined, such effects can be represented by appropriate constants, the values of which are then used to determine the value of a.

SUMMARY AND CONCLUSIONS

Numerical data pertaining to the spread of plant diseases from centers of infection to the above-ground parts of nearby susceptible hosts were compiled from the literature, and the rate at which the incidence of infection diminished with increases in distance from the source of inoculum examined. Some data collected by the present writers were also included. The diseases were of three categories with respect to the mode of inoculum dissemination: (1) Fungus diseases spread by means of air-borne spores. (2) A bacterial disease spread by means of wind-blown rain. (3) Virus and fungus diseases spread by insect vectors.

The gradients of infection that develop in the vicinity of sources of inoculum were compared with the pattern which in earlier studies appeared to characterize the aerial dissemination of spores. According to the results of these studies, for a distance of 15 feet at least from the source, the density, or number of air-borne spores passing through unit areas of planes perpendicular to the direction of the wind, diminished inversely proportional to the square of the distance from the source of inoculum, and the rate at which dispersion (the scattering of spores in dimensions perpendicular to the direction of the wind) increased was directly proportional to the distance from the source. Thus, the gradient of aerial spore density was described by the equation, $y = \frac{100}{x^2}$, where y is the density at x_2 and subsequent distance intervals expressed as a percentage of the density at x_1, or first distance interval. In cases where the disease resulted from infection by air-borne spores, the decrease in incidence of infection at increased distance from the source was described to a fair degree of accuracy by a modified form of this equation, *i.e.*,

$$y = \frac{100(1+a)^2}{(x+a)^2}. \qquad (3)$$

Apparently the constant a in this equation depends upon the conditions under which spore dissemination occurs, as well as upon the location of x_1 with respect to distance from the source.

Data pertaining to the spread of angular leaf spot of cotton by wind-blown rain, yellows of endive by leafhoppers, yellow dwarf of potatoes by leafhoppers, and Dutch elm disease by bark beetles, suggest that in such cases, also, the relationship between incidence of infection and distance from the source, in some respects, is similar to that occurring in the spread of diseases by air-borne spores.

An equation, employed by Gregory for the transfer of atmospheric properties by eddy diffusion and representing the dispersion of air-borne particles downwind from a point source, was examined. When normal values for the constants of this equation were employed, the resulting curve differed little from the curve given by equation 3 when $a = 0.21$. At low atmospheric turbulence or during short periods of observing spore dissemination, the exponent of x in the eddy diffusion equation is said to be at its minimum value, 1.24. Under such conditions the curve of the equation represents a less rapid decrease in incidence of disease with increased distance than does the equation 3 curve which corresponds most closely to it at locations near the source. Thus, the two types of curves diverge at some distance from the source.

Field results, therefore, will probably not reveal the small difference in relationships between the two equations except possibly during times of very low atmospheric turbulence, or when the period of effective dissemination is very short.

The major difficulty encountered in applying the eddy diffusion equation to disease gradient data arises from its failure to account for the combined effects of a large number of variables. The family of curves produced by equation 3 seems more applicable to the diverse conditions met with in the field.

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REACTIONS OF OAT VARIETIES AND SELECTIONS TO FOUR RACES OF LOOSE SMUT[1]

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Loose smut, *Ustilago avenae* (Pers.) Rostr., and covered smut, *U. kolleri* Wille (*U. levis* Magn.) are major diseases of oats in the United States causing an estimated average annual loss from 1935 to 1939 of 38 million bushels[3] of grain.

These smuts may be controlled either by treating the seed, before planting, with New Improved Ceresan or formaldehyde, or by growing varieties highly resistant to smuts.

Breeding of oat varieties resistant to smut is complicated by the number of physiologic races of loose and covered smut that are known and being found. Sometimes these new or previously unknown races become prevalent after varieties resistant to known races have been bred and distributed for commercial production.

The purpose of the investigation reported here was to determine the reactions of a number of varieties and promising advanced hybrid selections of oats to 4 races of loose smut that have been collected recently in Kansas.

REVIEW OF LITERATURE

Reed (6) demonstrated 29 physiologic races of loose smut and 14 races of covered smut from collections of oat smut made in 15 states in the United States, 9 countries, and 4 continents. Navarro, Markton, and Victoria were inoculated with all of the races and were resistant in all cases.

Navarro and Victoria were highly resistant to races of smut in the United States as shown by results from the Cooperative Uniform Oat Smut Nurseries conducted in 9 to 15 states each year during 1935 to 1939 (13). Neither of these varieties had any smut during this period, which represents over 50 station-year tests for each variety. Markton was resistant, averaging 0.23 per cent smut for 65 station years. Because of its high resistance to smut and also to most races of crown rust, Victoria was used extensively as a parent for breeding varieties of oats resistant to smut and rust (5). Markton was used extensively also as a parent and Navarro also was used (2 and 5). A race of covered smut has been reported, however, to which

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[3] U. S. Dept. Agr., Plant Disease Reporter. Supplement: 94, 1936; 100, 1937; 108, 1938; 118, 1939; 127, 1940.

Markton is intermediate in susceptibility. Smith and Bressman (10) inoculated seed of Markton, with hulls on and hulls off, with this race and obtained an average infection of 7 and 34 per cent smut, respectively. Unfortunately the collection of smut spores for inoculation by these investigators has been lost, thus precluding checking of any new races it may have contained.

In 1943, Reed and Stanton (7) demonstrated a new race of loose smut, collected in Oklahoma, to which Victoria and Fulghum were susceptible while Markton, Navarro, and Fultex were resistant. All of the 22 promising advanced selections of the cross Lee × Victoria, which were resistant to all races of smut prior to this time, were moderately to highly susceptible to this new race.

MATERIAL AND METHODS

The 4 races of loose smut[4] used in this study were collected in Kansas, although 2 of them were from fields planted with certified seed from other states. Race A was collected in a field of Kanota oats in Harper County in 1937 by J. O. Miller, Extension Plant Pathologist, Kansas State College. Fulghum and Monarch were susceptible to this race in 1940 after which the collection was propagated alternately on these 2 varieties. The inoculum used in 1945 was propagated on Monarch in 1944.

Race B was collected on an unknown variety of oats in Elk county in 1939 by J. O. Miller. Richland and Monarch Selection were susceptible to this race. The collection was propagated alternately on these two varieties. The inoculum used in 1945 was propagated on Monarch Selection in 1944.

Race C was collected by the writers in a test plot of Columbia oats in Sedgwick County, Kansas, in 1944. This plot had approximately 2 per cent of the panicles infected with smut. Certified seed of Columbia oats was obtained from a grower in Missouri for planting these plots.

Race D was collected in 1944 by the writers in a test plot of Fultex oats in Sedgwick County, Kansas, in the same field in which race C was collected. This plot had approximately one per cent of the panicles smutted. Certified seed of Fultex oats was obtained from a grower in Texas for planting these plots.

During the winter of 1944–45 the reactions of 9 smut-tester varieties and 18 other varieties to race D from Fultex were determined (Table 1). Seed with hulls off were inoculated with dry chlamydospores. Two replications of 125 seeds each of the 27 varieties were planted in a soil bed in the greenhouse. The temperature between planting and emergence was kept at 60° to 70° F. and the soil was maintained moderately dry. One hundred or more plants were obtained for each variety except for a few of the smut-tester varieties.

In the spring of 1945 the reactions to these 4 races of loose smut were studied on 12 smut-tester varieties, 21 named varieties, and 20 promising

[4] These four physiologic races of loose smut will be tentatively designated as A, B, C, and D. Eventually they will be numbered according to a standard classification.

hybrid selections grown in the 1945 Cooperative Uniform Spring Sown Red Oat Test. In addition, all of the oat strains in the advanced yield test at Manhattan were tested to determine their reactions to race D. The seed with hulls on were inoculated by the partial vacuum method described by Zade (15) and modified by Haaring (3) using a suspension of 0.5 g. of chlamydospores in 100 cc. of water. The seed was dried and planted the following day in 2 replications in the field at the rate of 5 g. to an 8-foot

TABLE 1.—*Reactions of oat varieties to a race of Ustilago avenae that attacks Richland and Victoria varieties (Race D). Greenhouse, Manhattan, Kansas, 1944–45*[a]

Variety	C.I. No.	Plants infected[b]
Smut-tester varieties		*Per cent*
Fulghum	708	3
Richland	787	90
Joanette	1762	100
Monarch	1876	100
Monarch Selection	1879	92
Gothland	1898	73
Victoria	2401	97
Red Rustproof	3212	0
Liberty Hull-less	845	100
Other varieties		
Brunker	2054	0
New Nortex	3422	0
Fulghum-Markton × Victoria-Richland	4140	0
Neosho	4141	0
Kanota	839	2
Navarro	966	2
Markton	2053	2
Bond	2733	2
Columbia	2820	12
Fulton	3327	26
Clinton	3971	26
Ventura	3989	53
Osage	3991	66
Boone	3305	91
Tama	3502	94
Vicland	3611	94
Cedar	3314	96
Fultex	3531	98

[a] Seed with hulls off were inoculated by dusting chlamydospores on the seed.
[b] Average of two replications, 4-foot rows.

row. Seed, with hulls off, of the same varieties were inoculated with dry chlamydospores of races C and D and then planted in the field in 2 replications at the rate of 2 g. to a 5-foot row.

The reactions of 8 smut-tester varieties, 18 named varieties, and 15 promising selections grown in the Cooperative Uniform Oat Smut Nursery were tested to a composite of several races of loose and covered smut which occur in Kansas, and to races A, C, and D. The composite did not include races A, B, C, and D nor the "Fulton" race of loose smut reported by Hansing *et al.* (4). Seed with hulls off were inoculated with dry chlamydospores

and then planted in the field in 2 replications at the rate of 1.5 g. to a 4-foot row.

In the field the soil temperature averaged 55° F. between planting and emergence. The soil was moderately dry the first 3 days after planting and then moderately moist for the following 4 days prior to emergence. Good stands were obtained for the group of varieties and hybrid selections planted with hulls on, while a fair stand was obtained for the rows planted with hulls off. A fair stand was obtained for some and a poor stand for others of the varieties and hybrid selections grown in the Cooperative Uniform Oat Smut Nursery.

In the greenhouse, percentages of infection were determined on the relative number of infected plants. In general, all of the panicles of a plant were smutted or all healthy. In the field, percentages of infection were determined on the relative number of infected panicles. Plants with some healthy panicles and some smutted panicles were common in the field.

EXPERIMENTAL RESULTS

Greenhouse Studies

Very high infection was obtained with race D in the greenhouse. Of the smut-tester varieties, Red Rustproof and Fulghum were resistant, averaging 0 and 3 per cent smutted plants, respectively (Table 1). The other smut-tester varieties were all susceptible, averaging from 73 to 100 per cent smutted plants. Victoria averaged 97 per cent smutted plants.

Brunker, New Nortex, Fulghum—Markton × Victoria—Richland, Neosho, Kanota, Navarro, Markton, and Bond were resistant, averaging from 0 to 2 per cent smutted plants. Columbia, Fulton, and Clinton were intermediate in susceptibility averaging from 12 to 26 per cent smutted plants. Ventura and Osage were susceptible, averaging 53 and 66 per cent smutted plants, respectively. Boone, Tama, Vicland, Cedar, and Fultex were very susceptible, averaging 91 to 98 per cent smutted plants.

Field Studies

Classification of physiologic races. Fulghum, Joanette, and Monarch were susceptible to race A, Gothland was intermediate in susceptibility, while all of the other 8 smut-tester varieties were resistant (Table 2). This race A represents a new race of loose smut distinct from any of Reed's 29 races (6). Reed did not have any race to which Fulghum and Monarch were susceptible while Canadian was resistant.

Richland, Canadian, Monarch Selection, Green Mountain, and Gothland were susceptible to race B, while the other differential varieties were resistant. This race B is similar to Reed's A–1 (6). It is commonly found where the variety Columbia is widely grown.

Richland, Canadian, Monarch, Monarch Selection, Green Mountain, and Gothland were susceptible to race C. Joanette was intermediate in susceptibility, while the other smut-tester varieties were resistant. This race C is somewhat similar to Reed's A–17.

TABLE 2.—*Reactions of oat varieties and selections to four races of Ustilago avenae in the field. Manhattan, Kansas, 1945*

Variety or selection	C.I. No.	Percentage infection by smuts[a]					
		Race A	Race B	Race C		Race D	
		Hulls on	Hulls on	Hulls on	Hulls off	Hulls on	Hulls off
Smut-tester varieties							
Fulghum	708	66	1	1	3	1	0
Richland	787	1	42	47	68	47	51
Canadian	1625	T[b]	90	88	98	92	98
Joanette	1762	50	0	17	22	78	92
Monarch	1876	82	0	62	77	92	98
Black Mesdag	1877	0	0	0	0	0	0
Monarch Selection	1879	T	86	78	84	80	84
Green Mountain	1892	0	82	78	92	75	94
Gothland	1898	12	86	68	78	11	26
Victoria	2401	0	0	T	0	85	88
Large Hull-less	3209	0	T	0	0	0	0
Red Rustproof	3212	0	0	0	0	0	0
Named varieties							
Markton	2053	T	0	0	0	0	0
Brunker	2054	0	T	0	0	0	0
New Nortex	3422	0	0	0	0	0	0
Neosho	4141	0	0	0	0	0	0
Navarro	966	0	0	0	0	T	0
Bond	2733	0	0	0	0	T	0
Kanota	839	69	2	2	4	T	1
Benton	3910	2	0	T	0	0	3
Clinton	3971	0	0	2	14	T	5
Marion	3247	0	3	1	3	T	6
Trojan	2491	0	6	5	16	3	6
Fulton	3327	0	3	4	4	4	5
Otoe	2886	0	2	42	46	2	9
Osage	3991	0	0	3	6	10	8
Ventura	3989	0	T	1	2	8	19
Columbia	2820	7	42	76	84	19	24
Boone	3305	0	0	0	0	69	79
Vicland	3611	0	0	0	0	72	84
Cedar	3314	9	0	0	0	74	83
Tama	3502	0	0	T	0	73	84
Fultex	3531	0	0	0	0	95	95
Hybrids							
Fulghum-Markton × Victoria-Richland	4140	0	0	0	0	T	T
do	4251	0	0	T	0	0	0
do	4335	0	0	0	0	72	87
do	4528	0	0	0	0	T	0
Fulton × Victoria-Richland	4338	T	0	2	7	4	8
do	4339	0	0	3	3	5	20
do	4340	0	0	1	2	6	17
do	4524	0	0	T	T	4	26
do	4525	T	T	0	0	87	89
Columbia × Victoria-Richland	4153	T	2	1	4	1	1
do	4246	0	2	10	21	1	3
do	4344	0	5	2	8	0	0
do	4345	0	2	1	0	0	0

TABLE 2—(*Continued*)

Variety or selection	C.I. No.	Percentage infection by smuts[a]					
		Race A	Race B	Race C		Race D	
		Hulls on	Hulls on	Hulls on	Hulls off	Hulls on	Hulls off
do	4346	0	T	0	0	61	72
do	4347	0	0	0	0	52	63
do	4349	0	1	1	0	51	56
do	4527	0	1	12	30	1	2
Victoria-Richland × Morota-Bond	4301	0	0	T	0	0	0
C.I. 4001 × C.I. 3644	4341	0	0	0	0	0	0
C.I. 4001 × C.I. 3842	4526	0	0	1	4	0	0

[a] Seed with hulls on were inoculated by partial evacuation. Seed with hulls off were inoculated by dusting the chlamydospores on the seed. Percentages represent averages of 2 replications. Hulls on = 8-foot rows, hulls off = 5-foot rows.
[b] T = 0.1 to 0.5 per cent infection.

Good infection was obtained with race D in the field for the highly susceptible varieties, although it was not so high as in the greenhouse. The infection for the varieties such as Ventura and Osage was not nearly so high in the field as in the greenhouse. Clinton and Fulton were intermediate in susceptibility in the greenhouse but had such low percentage of infection in the field that it was not possible to differentiate the relative susceptibility of similar varieties and hybrid selections from each other and from those which had high resistance.

Richland, Canadian, Joanette, Monarch, Monarch Selection, Green Mountain, and Victoria were susceptible to race D. Gothland was intermediate in susceptibility, while the other smut-tester varieties were resistant. The reactions of the smut-tester varieties demonstrate that race D is a new race of loose smut. It differs from the "Victoria" race which Reed and Stanton described as A–30 (7) in that Fulghum is resistant, while for their race Fulghum was susceptible.

In the Cooperative Uniform Oat Smut Nursery, similar infection was obtained for 8 smut-tester varieties to races A, C, and D (Table 3). For the Kansas composite of other races of loose and covered smut, Canadian, Monarch, and Fulghum were susceptible, with 93, 79, and 67 per cent smutted panicles, respectively. Gothland had 28 per cent smutted panicles, while the other smut-tester varieties had from 0 to 6 per cent smutted panicles.

Reactions of named varieties and hybrids. Kanota was susceptible to race A, with 69 per cent smutted panicles. All of the other varieties and advanced hybrid selections were resistant to this race, with from 0 to 7 per cent smutted panicles (Tables 2 and 3). Approximately 80 per cent of the oat strains had no smut.

Columbia was susceptible to race B, having 42 per cent of the panicles infected. The other oat varieties and hybrid selections inoculated with race

TABLE 3.—*Reaction of varieties and selections in Cooperative Uniform Oat Smut Nursery to a composite smut inoculum of loose and covered smuts and to physiologic races of loose smut in the field. Manhattan, Kansas, 1945*

Species, group, and variety	C.I. No.	Percentage infection by smuts[a]			
		Composite[b]	Race A	Race C	Race D
Smut-tester varieties					
Avena sativa					
Early black oats					
Monarch	1876	79	91	68	88
Black Mesdag	1877	6	0	0	0
Midseason white oats					
Canadian	1625	93	1	94	96
Gothland	1898	28	11	58	16
Avena byzantina					
Early red oats					
Fulghum	708	67	61	3	2
Columbia	2820	2	9	72	21
Midseason red oats					
Red Rustproof (Appler)	1815	0	0	0	0
Victoria	2401	0	0	0	90
Resistant varieties					
Avena sativa					
Early white oats					
Forvie	4164	0	0	0	26
Victoria-Richland × Morota-Bond	4301	0	0	0	2
Early yellow oats					
Cedar	3314	0	0	0	82
Clinton	3971	0	0	4	2
Benton	3910	0	0	0	0
D69 × Bond	4259	0	0	2	1
Mindo	4328	0	0	0	0
Midseason white oats					
Mission	2588	0	0	9	3
Markton × Victory	2591	0	0	0	0
Markton × Ligowa	3025	0	0	0	0
Markton × Ligowa	3633	0	0	0	0
Bonda	4329	0	0	0	0
Victoria-Richland × Bannock	4181	0	0	0	0
Victory × (Victoria-Richland × Markton-Victory)	4523	0	0	0	0
Midseason yellow oats					
Markton	2053	0	0	0	0
Bond × Rainbow	4331	1	0	0	1
Bond × D67	4327	0	0	3	2
Goldwin	4237	0	0	0	1
Stanton Strain 2	4390	0	0	0	81
Florilee	4060	0	0	0	86
Lee-Victoria × Fulwin	4316	0	0	0	92
Fulwin × Lee-Victoria	4383	0	0	8	....
Traveler	4206	0	0	0	96
Avena byzantina					
Early red oats					
Ventura	3989	0	0	4	8
Osage	3991	0	0	2	9
Neosho	4141	0	0	0	0
Columbia × Columbia-Navarro	4241	0	0	15	0
Columbia × Victoria-Richland	4522	0	7	15	14
Columbia × Victoria-Richland	4346	0	0	0	26
Fulgrain Strain 7	4389	0	0	0	38
Victorgrain Strain 5	4388	0	0	0	70

TABLE 3—(*Continued*)

Species, group, and variety	C.I. No.	Percentage infection by smuts[a]			
		Composite[b]	Race A	Race C	Race D
Midseason red oats					
Bond	2733	0	0	2	0
Red Rustproof[b] × Victoria-Richland	4385	0	0	0	0

[a] The hulls were removed before the seed were inoculated.
[b] Composite of several races of *Ustilago avenae* and *U. kolleri* which occur in Kansas. *U. avenae* races A, B, C, D, and "Fulton" were not included in this composite.

B averaged from 0 to 6 per cent smutted panicles (Table 2). Approximately 60 per cent were free from smut.

Columbia was susceptible to race C, averaging 80 per cent infected panicles. Otoe, generally considered as a moderately resistant variety, averaged 44 per cent smutted panicles when inoculated with race C. Trojan, Mission, and Clinton averaged 10, 9, and 7 per cent smutted panicles, respectively, with this race. Four of the 10 hybrids which had Columbia as one parent were intermediate in susceptibility, averaging from 15 to 21 per cent smut (Tables 2 and 3). Apparently a recombination of genetic factors occurred in these crosses to give this intermediate type of susceptibility. The other varieties and hybrid selections averaged from 0 to 8 per cent smutted panicles. Approximately 50 per cent of the varieties and hybrids were free from smut.

Of the commercial varieties grown in the United States, Fulghum and Kanota were susceptible to race A, Richland and Columbia to race B, and Richland, Columbia, and Otoe to race C. However, many of the new varieties and promising hybrid selections were susceptible to race D. Fultex (Fig. 1, A), distributed in Texas in 1940 as a smut-resistant variety (11), averaged 95 per cent smutted panicles. Boone, Cedar, Tama (Fig. 1, B), and Vicland, distributed as smut-resistant varieties in one or more north central states from 1940 to 1943 (5, 9, 11, and 12), were all susceptible, averaging from 74 to 78 per cent smutted panicles. In contrast Fultex, Boone, Cedar, Tama, and Vicland have averaged from 0.0 to 0.3 per cent smutted panicles in the Cooperative Uniform Oat Smut Nurseries (13 and 14). Florilee, a selection from a Lee × Victoria cross and distributed in Florida in 1944 (14), had 86 per cent smutted panicles, while Traveler, distributed in Arkansas in 1944 (14), had 96 per cent smutted panicles. Stanton Strain 2, Fulgrain Strain 7, and Victorgrain Strain 5 had 81, 38, and 70 per cent smutted panicles, respectively. Ventura, distributed in California in 1943 (1), and Osage, distributed in Kansas in 1945 (1 and 4), were susceptible in the greenhouse but moderately resistant in the field, averaging 12 and 9 per cent smutted panicles, respectively. Columbia and Forvic were intermediate in susceptibility to this race.

Culms of Ventura and Osage with smutted panicles were approximately

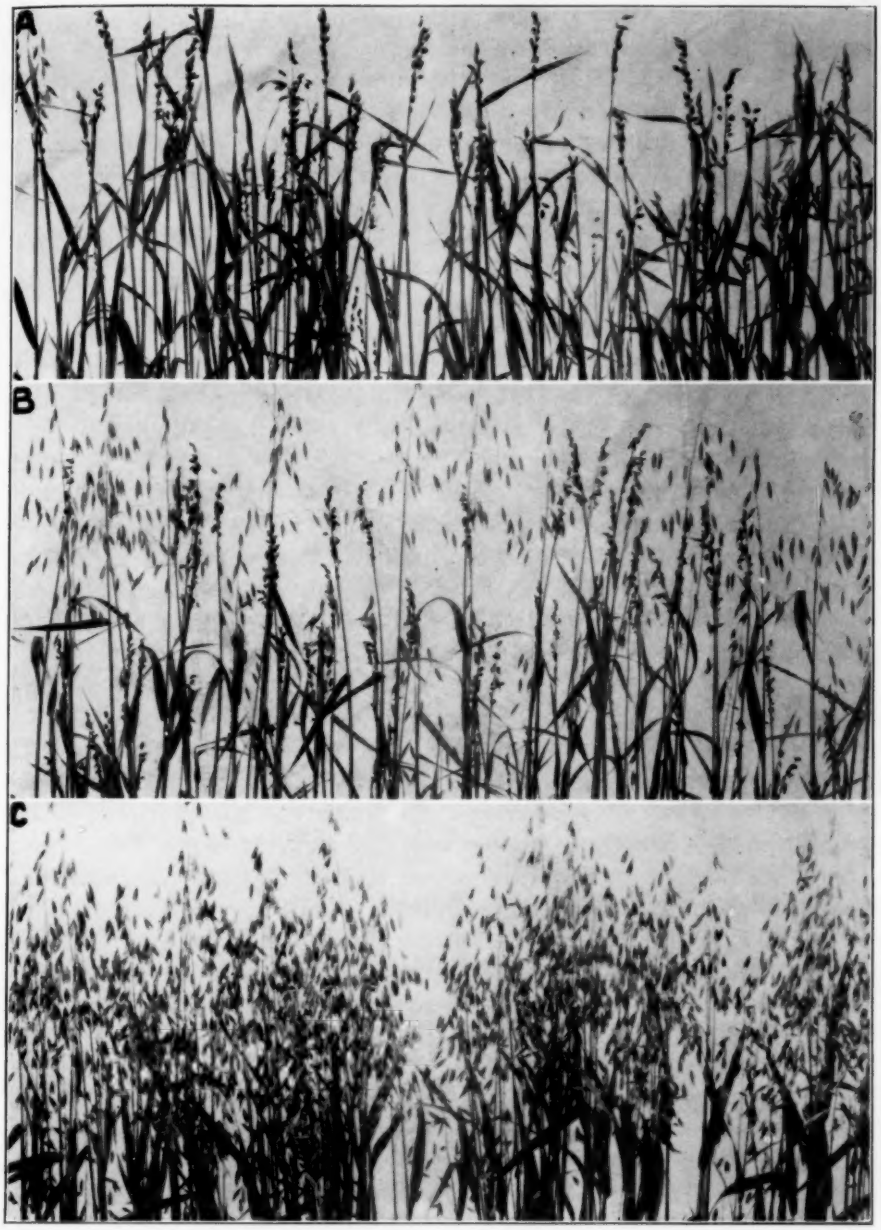

FIG. 1. Reaction of oat varieties to *Ustilago avenae* race D. A. Fultex, 95 per cent smutted panicles. B. Tama, 75 per cent smutted panicles. C. Neosho, 0 per cent smutted panicles.

40 per cent of the height of culms with healthy panicles. In contrast, culms of Boone, Cedar, Tama (Fig. 1, B), and Vicland with smutted panicles were approximately 90 per cent of the height of culms with healthy panicles.

Neosho (Fig. 1, C), distributed in Kansas in 1945 (1 and 4), was highly resistant. This variety has been highly resistant in other tests in Kansas and in the Cooperative Uniform Smut Nursery where it had an average of less than 0.1 per cent smut for 29 station years during 1943 and 1944 (14). It has yielded well in Kansas and in other states (1) and it is resistant to the common races of crown and stem rusts, but lacks resistance to stem rust races 8 and 10. Neosho is one of the few new commercial varieties with resistance to crown and stem rusts and as high or higher resistance to most races of smut as the old standard smut resistant varieties, Navarro, Markton, and Victoria.

Some of the hybrid selections were highly resistant while others were susceptible (Tables 2 and 3). Three selections from the cross Fulghum-Markton × Victoria-Richland were highly resistant while one was susceptible. The resistant characteristic probably came from the Fulghum-Markton parent, since both of these varieties were resistant to race D, while the Victoria-Richland parent was susceptible.

In the cross Fulton × Victoria-Richland, 4 selections were moderately resistant to intermediate in susceptibility to race D, having a reaction similar to Fulton, while one selection, C.I. 4525, was susceptible, averaging 88 per cent smutted panicles. C.I. 4525 or Kans. Sel. 41369 was highly resistant to the Fulton race of loose smut described by Hansing *et al.* (4). This would indicate that the reactions to race D and to the Fulton loose smut are probably determined by different genetic factors.

In the cross Columbia × Victoria-Richland, 5 selections were found to have more resistance to race D than Columbia (Table 2). This resistance probably was due to recombination of genetic factors. Three selections were susceptible, similar to the Victoria × Richland parent. When inoculated with race D approximately 30 per cent of the varieties and hybrid selections had no smutted panicles (Tables 2 and 3).

Out of 71 additional hybrid selections grown in the advanced yield nursery at Manhattan, 39 did not have any infection when inoculated with race D, 12 were resistant (trace to 10 per cent), 13 were intermediate in susceptibility (11–40 per cent), and 7 were susceptible (41–100 per cent).

DISCUSSION

The presence of a physiologic race of loose smut in the Southwest that attacks Richland and Victoria is of paramount importance because many of the new varieties of oats involving these strains as parents are widely distributed in the United States. As this race was introduced into Kansas on certified seed of Fultex oats from Texas, it may be assumed that it is already widely enough distributed that it can not be successfully eliminated by a seed treatment program. It may be only a matter of time until many of the new smut-resistant varieties now grown in the United States will be infected with this race so that the growers will have to treat their seed to control smut.

The occurrence of new physiologic races of oat smut is an important problem that confronts investigators who are breeding disease-resistant varieties of oats. In 1919 Kanota, a Fulghum type oat, was distributed in Kansas (1, 4, and 8) as a smut-resistant variety. It soon became a popular variety and its acreage increased rapidly. However, physiologic races of loose smut existed in the southern oat-growing area of the United States to which Kanota was susceptible. By 1928 these races of smut had spread in Kansas to such an extent that Kanota could no longer be considered as a resistant variety in the State (4).

In 1939, Fulton, a selection from a cross, Fulghum × Markton, was distributed in Kansas (4 and 11) as a smut-resistant variety. Fulton was resistant to the prevalent races of smut in the United States, averaging 1.29 per cent infected panicles for 43 station-year tests in the Cooperative Uniform Oat Smut Nursery (13). However, with artificial inoculation it was intermediate in susceptibility to a new physiologic race of loose smut (4) that had existed as a slight mixture in a few of the Kansas collections of oat smut. This new race of smut was distributed with Fulton seed to growers of certified seed and by 1945 was distributed fairly well in Kansas. Fortunately Fulton oats was moderately resistant to this race of loose smut in the field. Since 1939, most of the fields of this variety have had none or only a trace of smut. However, as high as 10 per cent smut has been recorded in a few fields.

No smut was observed in the increase plots of Osage and Neosho in 1944. However, the seed was treated with New Improved Ceresan before it was distributed to growers of certified seed in 1945. It is desirable that growers of certified seed of these varieties continue to treat their seed.

Oat smuts are seed-borne diseases and are distributed principally on the seed. Growers of certified seed and other producers are partly responsible for the spread of new races of smut. It may be possible to slow down or even stop the distribution of a new race of smut if the seed of new varieties is treated properly to control smut. Another way of spreading new races of smut is through the interchange of new hybrid material among plant investigators in different states.

New varieties of oats that are resistant to known races of smut are bred by plant breeders and plant pathologists. Races of smut, however, exist or develop by hybridization or mutation some place in the country. These gradually spread until the new variety, after a decade or two, can no longer be considered a resistant variety.

In the meantime the plant breeder and plant pathologist may have a new variety ready for distribution, which is resistant to the old races of smut as well as to the new ones. In this way the research work on breeding disease-resistant varieties has generally managed to keep abreast of natural production of new races of smut. On the other hand, if seed dealers would treat all of their seed prior to selling, the problem of smut-resistant varieties becoming susceptible to new races of smut in the field

would be attacked from two angles. Progress in the development of new smut-resistant varieties of oats would probably keep farther ahead of the natural development and distribution of new races of smut than when attacked chiefly by the plant research workers alone.

SUMMARY

The four physiologic races reported in this investigation were collected in Kansas. Two of the races (A and D) were distinct from those reported by other investigators. Race A is characterized by the susceptibility of the varieties Fulghum and Monarch and the resistance of Canadian. Race D is characterized by the susceptibility of Richland and Victoria and the resistance of Fulghum.

Of the commercial varieties, Kanota and Fulghum were susceptible to race A. Richland and Columbia were susceptible to race B. Richland, Columbia, and Otoe were susceptible to race C.

Richland, Boone, Cedar, Tama, Fultex, Vicland, Florilee, and Traveler were susceptible to race D. Columbia and Forvic were intermediate in susceptibility. Ventura and Osage were susceptible in the greenhouse but intermediate to moderately resistant in the field. Benton, Clinton, Fulton, Mission, and Goldwin had some resistance to this new race of loose smut.

Black Mesdag, Large Hull-less, Red Rustproof, Navarro, Markton, Brunker, Bond, New Nortex, Neosho, Bonda, Mindo, and several promising hybrid selections were highly resistant to all 4 races.

Neosho appears promising as a parent for oat crosses. It is highly resistant to smut, resistant to the common races of crown and stem rusts and has desirable agronomic characters. Neosho was distributed in Kansas in 1945.

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SEED AND SEEDLING INFECTION OF BARLEY, BROMEGRASS, AND WHEAT BY XANTHOMONAS TRANSLUCENS VAR. CEREALIS[1]

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Little is known concerning the seed and seedling infection of barley, bromegrass, and wheat by *Xanthomonas translucens* var. *cerealis*. Natural seed infection of barley and wheat has been reported, but no infection of bromegrass seed by this bacterium has been observed except for some lesions on the glumes enveloping the seeds (9). The seed-borne nature of *Xanthomonas translucens* on barley was described first by Jones, Johnson, and Reddy in 1916 (5). Seedling infection of barley from naturally infected seed was later described by Smith, Jones, and Reddy in 1917 (6), and the same process was postulated for wheat in 1919 (8). Jones, Johnson, and Reddy in 1917 (6) described barley seedling infection from barley seed naturally infected with *X. translucens*. In 1936, Bamberg (1) was unable to find a single infected wheat seedling in the field or in the greenhouse from seed naturally infected with *X. translucens*. In the same year, Hagborg (4) obtained 81 per cent infected seedlings from wounded wheat seeds which were infested with *X. translucens* and planted in the soil.

In 1941 Galachian (2) infected wheat seedlings with barley and wheat isolates of *Bacterium atrofaciens* by wounding the young plumules with a needle carrying the bacteria. In the same year Gorlenko (3) obtained 42 per cent diseased wheat seedlings by infesting wheat seeds with *Xanthomonas translucens* var. *cerealis* following an initial needle wounding of the seeds close to the embryo. Although Gorlenko and Hagborg showed that wounded infested seeds resulted in a high percentage of diseased seedlings they, and the earlier workers, did not define the course which the pathogen followed in moving from the seed surface to the aerial portions of the plant.

The purpose of this study was two-fold; first, to study the influences of races of *Xanthomonas translucens* var. *cerealis* on the growth of seeds and seedlings of barley, bromegrass, and wheat; and second, to trace the movement of this organism into the embryo and follow the spread of the pathogen from the seed up into the primary leaf.

MATERIALS AND METHODS

The barley and wheat varieties used were obtained from the Agronomy Farm at Iowa State College, while the strains of bromegrass were obtained

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from the Soil Conservation Service at Ames.[3] As far as possible disease-free seeds were used. Prior to infestation all seeds were immersed for ten minutes in a 15 per cent solution of commercial clorox,[4] and rinsed several times in distilled water.

The seeds were wounded by rupturing the testa covering the embryo with a dissecting needle, under a binocular. Four races of *Xanthomonas translucens* var. *cerealis*, whose history and performance were already known (9), were used because they were isolated from the same hosts employed in the present trials. All bacterial races were maintained on nutrient agar.

For seed infestation four 24-hour beef-peptone agar cultures of each race were used. The culture tubes were half filled with water and the bacteria were scraped into the water with a nichrome wire loop. The suspensions from 4 tubes of any one race were emptied into a single flask containing the seeds to be infested. The soaking seeds were then subjected to a partial vacuum by a water vacuum pump for 5 minutes and allowed to soak in the suspension for one hour or more depending upon the experiment. Usually the seeds were planted on sterilized moist filter papers in 9-cm. Petri dishes. In a few tests infested seeds were also planted in steamed compost soil in the greenhouse. The seedlings were usually examined for germination and disease seven days after plating in the laboratory.

THE INFLUENCE OF XANTHOMONAS TRANSLUCENS VAR. CEREALIS ON THE GERMINATION AND SEEDLING INFECTION OF NONHULLED AND HULLED BARLEY SEED

While Hagborg (4) and Gorlenko (3) studied the pathogenicity of *Xanthomonas translucens* on wheat by infesting wounded seeds in bacterial suspension, Kingsolver (7) found that hulled oat seed without intentional wounding gave a higher percentage of infection than nonhulled oats when soaked in suspensions of *Pseudomonas coronafaciens*. This finding aroused speculation as to what might happen with barley and bromegrass which are well fortified with hulls.

At the outset, the pathogenicity of races 1, 4, 5, and 6 of *Xanthomonas translucens* var. *cerealis* was tested on nonhulled barley in three experiments. No difference in germination and seedling infection was noted between the infested and the noninfested seeds. In further tests different lots of hulled and nonhulled Spartan barley were infested with race 6 by soaking in a bacterial suspension for one, two, three, and twenty-four hours. No differences in germination were noted in the hulled or nonhulled seeds, either infested or noninfested. The percentages of diseased seedlings in the infested nonhulled series were 12, 17, 17, and 50 for 1, 2, 3, and 24 hours' soak periods, respectively, while in the hulled series for the same periods the percentages were 54, 57, 57, and 67. The removal of the barley hulls had the

[3] The author wishes to extend his gratitude to Dr. H. C. Murphy for supplying the seeds of the small grains. Also, it is a pleasure to acknowledge the assistance of Mr. M. L. Heath of the Soil Conservation Service at Ames in supplying the bromegrass seeds.

[4] Commercial clorox is a 1.5 per cent solution of sodium hypochlorite.

same effect on seedling infection as increasing the period of soak. In two additional experiments hulled and nonhulled Glabron barley were used. In the first of these the seeds were soaked for 1, 5, 10, 15, 20, and 25 hours, while in the second the seeds were soaked for 1, 5, 10, 24, and 58 hours. Again no influence on germination was noted for any of the treatments. In the infested series the percentage of infected seedlings was higher with the hulled seeds than with the nonhulled as in the earlier trials. With hulled seeds the number of infected seedlings was the same through the range of soaking periods. On the other hand, with the nonhulled seeds the number of diseased seedlings was greater with the longer soaking. In these trials the seedlings were infected without deliberate wounding of the seed. Of course, some wounding may have occurred in the process of removing the hulls.

THE INFLUENCE OF TEMPERATURE ON EMERGENCE AND SEEDLING INFECTION FROM SEED SOAKED IN DIFFERENT STRAINS OF XANTHOMONAS TRANSLUCENS VAR. CEREALIS

The influence of temperature on the pathogenicity of the organism on hulled and nonhulled seed was desirable to know. Also, with hulled seed it was essential to determine the influence of the temperature during germination on the seeds and seedlings. In a preliminary experiment with hulled barley infested with races 1 and 6 of *Xanthomonas translucens* var. *cerealis* and held at 15° C. and 25° C., no difference in germination was found and the percentages of infected seedlings were the same at both temperatures.

To determine whether or not the temperature during seed infestation influenced emergence and the seedling infection, nonhulled seeds of Glabron and Velvet barley were infested by soaking in suspensions of race 6 of *Xanthomonas translucens* var. *cerealis* for 24 hours at 10°, 15°, 20°, 25°, 30°, and 35° C. Corresponding lots of seed were soaked simultaneously at the different temperatures in distilled water. The seeds were then planted in potted steamed soil and were placed in a dark incubator at 25° C. When the young seedlings were 5 cm. high the emergence was recorded and the pots containing the seedlings were transferred to the greenhouse. Two days later the diseased seedlings were counted. (See table 1.)

The emergence of the two varieties was erratic in both infested and noninfested seed lots. At 30° and 35° C. a definite reduction of emergence was obtained in the infested and noninfested series. Seedling infection from the infested seeds occurred at all six temperatures. The total number of infected plants for all temperature exposures was 34 with Velvet and only 24 with Glabron barley, which is only 3 and 2 per cent, respectively, of the total number infested. Apparently during the period of soaking the temperature had no influence on the penetration of the bacteria beneath the hulls since the percentages of infected seedlings for both varieties were low at all temperatures.

The influence of temperature on emergence and seedling infection during the germination of seeds infested with races 1 and 6 of *Xanthomonas trans-*

TABLE 1.—*The emergence and seedling infection of two barley varieties soaked in water and in a suspension of race 6 of Xanthomonas translucens var. cerealis for 24 hours at six different temperatures*

Temperature, degrees C.	Velvet				Glabron			
	Water		Race 6		Water		Race 6	
	Emer. Pct.[a]	Inf. Pct.[a]	Emer. Pct.	Inf. Pct.	Emer. Pct.	Inf. Pct.	Emer. Pct.	Inf. Pct.
10	90	0	92	2	91	0	91	2
15	81	0	71	6	86	0	80	2
20	90	0	72	5	90	0	84	3
25	73	0	88	3	86	0	91	1
30	74	0	66	3	36	0	74	5
35	13	0	2	0	15	0	63	1

[a] Each percentage represents the emergence from four replications of 50 seeds in each. Infection percentage was based on the number of seedlings that emerged.

lucens var. *cerealis* was studied. The seeds were infested by soaking them six hours at room temperature in suspensions of two races of the bacterium. The seeds were then planted, as before, in potted steamed soil and placed in the incubators until the seedlings were 5 to 7 cm. high, when the pots were transferred to the greenhouse. At this time emergence was recorded and seven days later seedling infection was recorded (Table 2). The emergence of the controls was at a high level, between 78 and 91 per cent, at temperatures of 10–25° C. and fell off slightly at 30–35° C. The emergence of the seeds infested with race 6 was lower than the controls at corresponding temperatures, except at 35° where there were no differences. The emergence percentage obtained using this race was the same at all temperatures except 30° C. where a reduction occurred. With race 1 the emergence decreased in a regular manner with increasing temperatures, from a high of 78 per cent at 10° C. to a minimum of about 12 per cent at 30° to 35° C. Therefore, there was less reduction of emergence in comparison to the controls at the lower than at the higher temperatures.

Seedling infection was obtained with both races of *Xanthomonas translucens* var. *cerealis*. The percentage of infected seedlings obtained with

TABLE 2.—*Emergence and seedling infection at six temperatures of hulled Glabron barley seeds infested with two races of Xanthomonas translucens var. cerealis*

Temperature, degrees C.	Water		Race 1		Race 6	
	Emer. Pct.[a]	Inf. Pct.[a]	Emer. Pct.	Inf. Pct.	Emer. Pct.	Inf. Pct.
10	89	0	78	6	69	13
15	78	0	65	12	60	10
20	91	0	50	22	63	16
25	78	0	31	16	58	14
30	67	0	12	33	47	15
35	61	0	13	77	63	14

[a] Each emergence percentage represents the emergence from 4 replications with 25 seeds in each. The infection percentage is based on the number of seedlings that emerged.

race 6 was approximately 14 at all temperatures and no significant differences due to temperature were noted. With race 1 the percentage of infected seedlings was generally greater as the temperature at which the seedlings were grown was higher. Thus the percentage of infected seedlings ranged from a low of six at 10° C. to a high of 77 at 35° C.

THE RELATION OF SEED INJURY TO GERMINATION AND SEEDLING INFECTION

The trials on nonhulled and hulled barley suggested that if the bacteria were allowed closer contact with the embryo they would penetrate the growing seedlings. Whether or not the pathogen entered the developing embryo was not known because the germinating seeds had not been killed. Hagborg (4) and Gorlenko (3) obtained high percentages of infected wheat seedlings by wounding and inoculating the seeds. These workers reported that wounding the seeds reduced the germination. In order to determine the influence of the bacteria on wounded seeds, trials were made with wounded barley, bromegrass, and wheat infested with different races of *Xanthomonas translucens* var. *cerealis*.

Barley Seed Wounded and Infested with Xanthomonas translucens var. cerealis race 6

In the first trial two samples of Peatland barley of 20 seeds each were hulled and wounded; two other samples were hulled only. Similar lots of Spartan barley were also prepared. Using the two varieties, one sample of the wounded seed and one sample of the hulled seed were soaked in suspensions of *Xanthomonas translucens* var. *cerealis* race 6, while the other samples were soaked in water. The seeds were planted on moist filter paper in sterile Petri dishes. Germination was counted three days after planting, when the diseased plumules ceased to elongate. The short plumules were

TABLE 3.—*Germination and seedling infection of two varieties of hulled barley artificially wounded and nonwounded soaked 20 hours in water and a suspension of race 6 of Xanthomonas translucens var. cerealis (20 seeds per plate)*

Barley variety and trial number	Water				Race 6			
	Hulled		Hulled, wounded		Hulled		Hulled, wounded	
	Germ. Pct.	Inf. Pct.[a]	Germ. Pct.	Inf. Pct.	Germ. Pct.	Inf. Pct.	Germ. Pct.	Inf. Pct.
Peatland								
1	90	0	90	0	55	54	15	100
2	95	0	90	0	90	17	85	77
3	95	0	80	0	90	11	65	77
Spartan								
1	70	0	60	0	80	37	20	100
2	100	0	85	0	100	20	90	61
3	80	0	85	0	80	19	80	69

[a] Percentage infection is based on the number of seedlings that developed.

frequently browned and appressed to the kernel. Final counts of diseased seedlings were recorded after seven days when the first foliage leaf had elongated sufficiently to have evidence of infection (See table 3).

In the control series the wounded and nonwounded hulled seeds of both varieties germinated well and the seedlings were healthy and vigorous. No significant reduction in germination attended the wounding of hulled seeds. In the series infested with bacteria the emergence of the nonwounded, hulled seeds was moderately reduced only in the first trial with Peatland barley, but not in the second or third trials. There was no reduction in germination with Spartan barley. With wounded hulled seed of both varieties there were marked reductions in emergence only in the first trial but generally none in the other two except for moderate reduction with Peatland barley in the third trial.

The percentage of diseased seedlings in all the trials of both varieties was much greater with the wounded, hulled seeds than with the nonwounded, hulled seeds. The occurrence of seedling infection in the nonwounded, hulled seed which was infested would suggest that wounding occurred during the hulling. An examination of hulled seeds through the binocular revealed that either the testa or the embryo were injured in many of the seeds.

Symptoms were observed on the young seedlings. The plumules that were not over 1.5 to 2 cm. long became yellow and water-soaked. On the coleoptile there were light brown lesions. Except for these inconspicuous lesions on the coleoptile some plumules appeared healthy. Later the lesions appeared on the first foliage leaf as it emerged from the coleoptile.

Bromegrass Seed Wounded and Infested with Xanthomonas translucens var. cerealis race 6

Preliminary experiments demonstrated that hulled bromegrass seed was more susceptible to *Xanthomonas translucens* var. *cerealis* than nonhulled seed. Therefore, three experiments were designed to determine if wounding increased the susceptibility of the germinating seeds to the bacteria. In another trial bromegrass seeds were hulled, divided into lots of 50 seeds, and wounded as before. The lots were then divided into two, 25 seeds each. One lot was soaked in water and the other was soaked in a suspension of the bromegrass pathogen. Twenty-five nonhulled and hulled seeds were included as checks against the hulled, wounded seeds. The germination and seedling infection were recorded five days following infestation. (See fig. 2.) The germination was the same in the nonhulled and hulled seeds in the controls. The germination of the hulled, wounded seed was reduced approximately 40 per cent, which indicated that the young embryos of the noninfested seeds may have been injured. Infestation reduced germination of nonwounded, hulled bromegrass seed and reduced it even more with wounded, hulled bromegrass seed. The lower germination in the nonhulled and hulled infested bromegrass seeds in comparison with the controls suggested that the bacteria had some influence on germination.

The infected seedlings were readily distinguished by the water-soaked lesions on the leaves. The percentages of infected seedlings based on the emergence were 4, 13, and 60 for the nonhulled, hulled, and hulled and wounded lots, respectively. There was no infection in the controls. Wounding the seed by splitting the testa favored the pathogen in its development in the seedling. Since the seedlings from nonhulled seed were infected, presumably the pathogen penetrated the hulls of the seed and contacted the embryo or the bacteria from the hulls contaminated the emerging coleoptiles.

Wheat Seeds Wounded and Infested with Races 1, 4, 5, and 6 of Xanthomonas translucens var. cerealis

Wheat, which in preliminary trials had been unscathed by any of the test cultures, was wounded and soaked in suspensions of four races of *Xanthomonas translucens* var. *cerealis*. Nonwounded seeds were similarly treated. After soaking for 20 hours, the seeds were drained and planted in sterile Petri dishes or steamed soil. Control lots were soaked in water. Five trials were made at 2-day intervals, three in Petri dishes and two in steamed soil. Records were taken after seven days.

Germination was reduced in the wounded infested series. Races 1 and 6 caused the greatest reduction in germination. The embryos of some seeds were covered by a yellow bacterial mass. None of the four strains of bacteria seemed to attack the embryos of the nonwounded seeds and no differences were obtained in the percentage germination of the control and of the infested seeds.

As in the barley and bromegrass trials, the percentages of infected seedlings were greater with wounded than with nonwounded seeds. With bromegrass the difference between the two was very much greater than with the other crops. Race 6 was the least pathogenic. Races 4 and 6 produced less seedling infection than 1 and 5. In the soil the percentages of infection were 50, 40, 56, and 40 for the four races used. Seedling development was also influenced by the pathogen. For example, the average plumule length for the eight seedlings in the first trial of wounded seeds infested with race 5 was 2.6 cm., while the plumule length in the check was 7.0 cm.

THE RESPONSE OF NONWOUNDED SEEDS SOAKED IN SUSPENSIONS OF XANTHOMONAS TRANSLUCENS VAR. CEREALIS AND PLANTED IN SOIL

Laboratory trials with seeds soaked in a bacterial suspension showed that such infested seeds gave rise to infected seedlings in Petri-dish plantings. This raised the question as to what might happen when such infested seeds were planted in the soil. To study this problem an experiment was designed to determine the influence of races 1 and 6 on emergence and seedling disease with hulled and nonhulled barley seed. Lots of 200 seeds each were soaked in bacterial suspensions of races 1 and 6, and in water as control. After one hour of soaking under partial vacuum the seeds were planted, 40 seeds per

pot, in 4-inch pots of steamed soil. Five replicate pots for each of the twelve treatments were planted and randomized as complete blocks on the greenhouse bench. The emergence counts were made after seven days and are presented in figure 1.

An analysis of the data indicated a highly significant reduction in emergence of hulled seed due to bacterial seed infestation. No significant differences were noted between the two races. The emergence of nonhulled seed was unaffected by bacterial infestation. The hulls seemed to protect the seed from bacterial injury. With removal of the hulls, better emergence of the seed was obtained in the control series than when the hulls were left on.

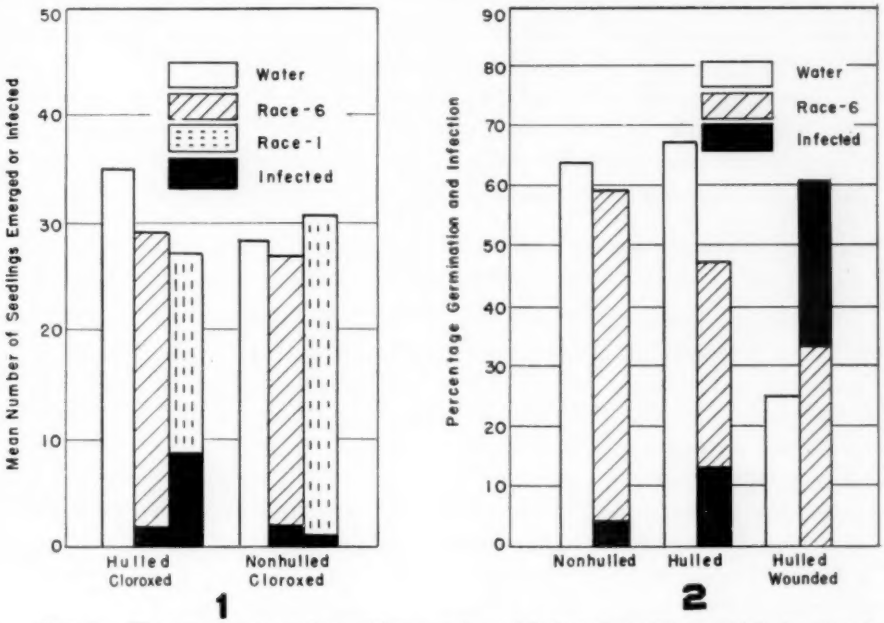

FIG. 1. The emergence and resulting number of infected seedlings which developed from hulled and nonhulled cloroxed barley seed soaked in suspensions of two races of *Xanthomonas translucens* var. *cerealis*. Seeds planted in steamed soil.

FIG. 2. The percentage germination and infected seedlings which developed from wounded and nonwounded bromegrass seeds soaked in a suspension of race 6 of *Xanthomonas translucens* var. *cerealis*.

The numbers of diseased plants recorded ten days following planting are in figure 1. The results indicated (1) seedling infection with both races of *Xanthomonas translucens* var. *cerealis;* (2) a greater number of plants infected with race 1 than with race 6; (3) a greater number of plants infected by both races when hulled seed was used. These infection results were in agreement with those obtained in the Petri-dish trials.

In a further experiment four races of the organism were compared. The nonhulled seeds were used and infested as before. The seeds were planted in potted steamed soil in four replications and randomized within a moist chamber where the temperature ranged from 15° C. at night to 25° C. dur-

ing the day. After seven days the pots were transferred from the chambers to the greenhouse bench and emergence was recorded. Seven days later notes were taken on the number of diseased seedlings.

A variance analysis of the data revealed a highly significant decrease in emergence due to the races 1 and 4, but not to races 5 and 6. All of these races induced low percentages of seedling infection, ranging from three with race 1 to six with race 5. Despite the low incidence of disease the experiment showed that infected seedlings could be obtained by planting infested seed in steamed soil in the greenhouse.

Wheat and bromegrass seeds also were tested with the same four races. None of the races influenced emergence of these grasses although a few seedlings were diseased in each of them. All races caused some wheat seedling infection while only race 6 infected bromegrass seedlings.

The small number of infected seedlings in these soil tests constituted further evidence that wounding favors infection. The data from the soil experiments substantiate the findings of the laboratory trials that wounding favors the entrance of the pathogen into the developing seedling.

THE MOVEMENT OF XANTHOMONAS TRANSLUCENS VAR. CEREALIS INTO THE SEED AND LEAVES OF THE DEVELOPING SEEDLING

Although Hagborg (4), Galachian (2), and Gorlenko (3) showed that diseased seedlings developed from wounded, infested wheat seeds, they did not attempt to explain how or where the bacteria entered the seed and the developing seedling. Two courses are open; either the pathogen contaminates the developing coleoptile as it emerges from beneath the hulls and subsequently passes through it to reach the first leaf or the pathogen penetrates beneath the hulls and testa of the seed and enters the young developing embryo. If the latter method is followed there must be a point in the embryo through which the bacteria would pass to reach the primary leaves of the seedling.

The present studies using nonhulled, hulled, and wounded hulled seeds have shown that high percentages of infected seedlings were obtained only when the seeds were wounded. When the testa over the embryo was split to allow for passage of the bacteria to the embryo, high percentages of the growing seedlings developed infected coleoptiles within 48 hours after infestation or had lesions on the first foliage leaves upon the leaf's emergence from the coleoptile. These facts indicated that the bacteria penetrated the coleoptile at some point soon after they were in contact with the tissues, since the symptoms were evident so soon after germination. Sections of diseased plumules present evidence to support this contention.

The symptoms on barley, bromegrass, and wheat seedlings were remarkably alike. Many diseased seedlings, 48 to 72 hours old, were examined with the binocular. The first apparent symptoms on plumules 3–5 cm. long were yellowing, distortion, and deformation of the coleoptile. Long, water-soaked, sunken streaks were on some coleoptiles. These streaks which were apparent

FIG. 3. Diseased foliage leaf of a barley seedling with water-soaked, translucent areas. Note the bacterial exudate.

before the foliage leaf had burst through the coleoptile, became brown and later split, thereby exposing the yellow water-soaked inclosed foliage leaf. Severely diseased plumules soon collapsed and died, either before or after emergence from the coleoptile. In less severely infected plumules the first foliage leaves emerged from the coleoptile and continued to develop. Scattered translucent areas developed along the unfolded leaves and these areas later enlarged into yellow-green, water-soaked streaks (Fig. 3). These streaks were clearly visible on barley and wheat, but were difficult to see on the narrow leaves of bromegrass. As the brome leaves increased in size, however, the streaks became clearly perceptible.

Free-hand sections cut from the base of the diseased plumule were studied under the microscope. Bacteria could be seen streaming out of the cut ends of the sections, but it was not always possible to determine the exact point of egression. Masses of bacteria and cell débris exuded from the tissues of the coleoptile and from the vicinity of the first foliage leaf. There were no lesions at the tip of the plumule nor was there any bacterial streaming from sections made at the tip.

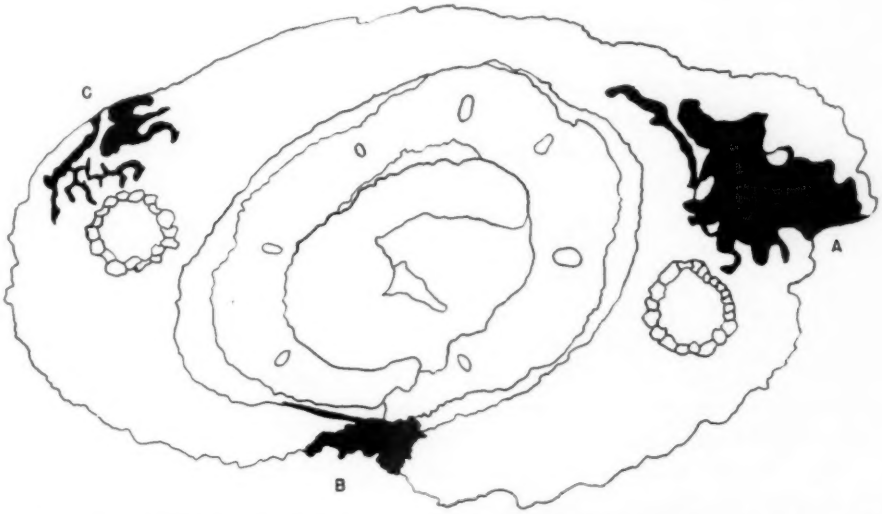

FIG. 4. A cross section of a barley plumule showing the lesions caused by race 6 of *Xanthomonas translucens* var. *cerealis*. The shaded areas indicate the points of entrance and the spread in the tissues. At A and C the bacteria were in the coleoptile while at B the parasite reached the first foliage leaf.

Plumules from which the free-hand sections had been cut in the preliminary diagnosis were killed in formalin-acetic-alcohol saturated with mercuric chloride. The mercury was added to prevent the leaching of the bacteria from the tissue. The tissues were run through the butyl alcohol series, embedded, and stained.

The sections were examined under the microscope for the location of lesions and the organism in the tissues. In these lesions there were masses of bacteria in the intercellular spaces extending from the outer to the inner surface of the coleoptile (Fig. 4). The first foliage leaf lying against the inner wall of the coleoptile was severely parasitized and partially disintegrated. The tip of the second foliage leaf abutting the first leaf was entirely destroyed and the space filled with débris and bacteria. The third leaf lying inside the second leaf was not invaded. The pathogen was intercellular in the parenchymatous tissues and it was never observed inside the endodermis.

There was no evidence that the bacteria invaded the seedling except through the coleoptile. Stomates were observed along the coleoptile and these served as natural openings for the entrance of the bacteria. Penetration was not observed in the root tissue or in the scutellum area. Primary infection presumably occurred through natural openings or wounds in the coleoptile either while in the embryo or in the early stages of plumule elongation. Presumably, the pathogen was carried up in the aerial parts of the seedling when the first foliage leaves emerged from the elongating infected plumules.

SUMMARY

Seed and seedling infection of barley, bromegrass, and wheat was studied with seeds infested with races of *Xanthomonas translucens* var. *cerealis*. The factors studied were duration of period during which the seeds were soaked in bacterial suspension, hulling and wounding the seeds, and the temperature during seed infestation and during seed germination and growth. The localization and the penetration of the bacteria into the developing plumule were studied also.

Hulled barley seed soaked for one hour in bacterial suspension produced 54 per cent infected seedlings while the nonhulled seeds which had been soaked in bacterial suspension for the same period produced only 12 per cent diseased seedlings. A 24-hour soak was necessary for the nonhulled seeds to yield 50 per cent diseased seedlings.

The temperature during seed infestation of nonhulled barley seed was not a factor influencing seed germination or seedling disease. The temperature during seed germination and seedling development influenced emergence and seedling infection when seeds were infested with race 1 of *Xanthomonas translucens* var. *cerealis*.

Infection of barley, bromegrass, and wheat seedlings was facilitated by rupturing the testa covering the embryo prior to seed infestation. Diseased seedlings were obtained from infested hulled barley and bromegrass seeds that were planted in the soil. A few diseased seedlings also developed from nonwounded infested wheat seeds.

Xanthomonas translucens var. *cerealis* penetrated through wounds in the pericarp covering the embryo, as demonstrated by the greater percentage of infected seedlings obtained from wounded seeds.

Infection of the plumule occurred through wounds or stomata on the coleoptile, spread rapidly through these tissues, and finally reached the inclosed foliage leaves. Since the first foliage leaf was adjacent to the coleoptile it became infected before emerging from the coleoptile. In this way, the infected leaf by elongation carried the bacteria into the aerial parts of the seedling. The initial symptoms were water-soaked streaks on the primary leaf.

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IDENTITY AND KNOWN DISTRIBUTION OF *ELSINOË PIRI* IN WASHINGTON AND OREGON

ANNA E. JENKINS, M. J. FORSELL AND L. W. BOYLE

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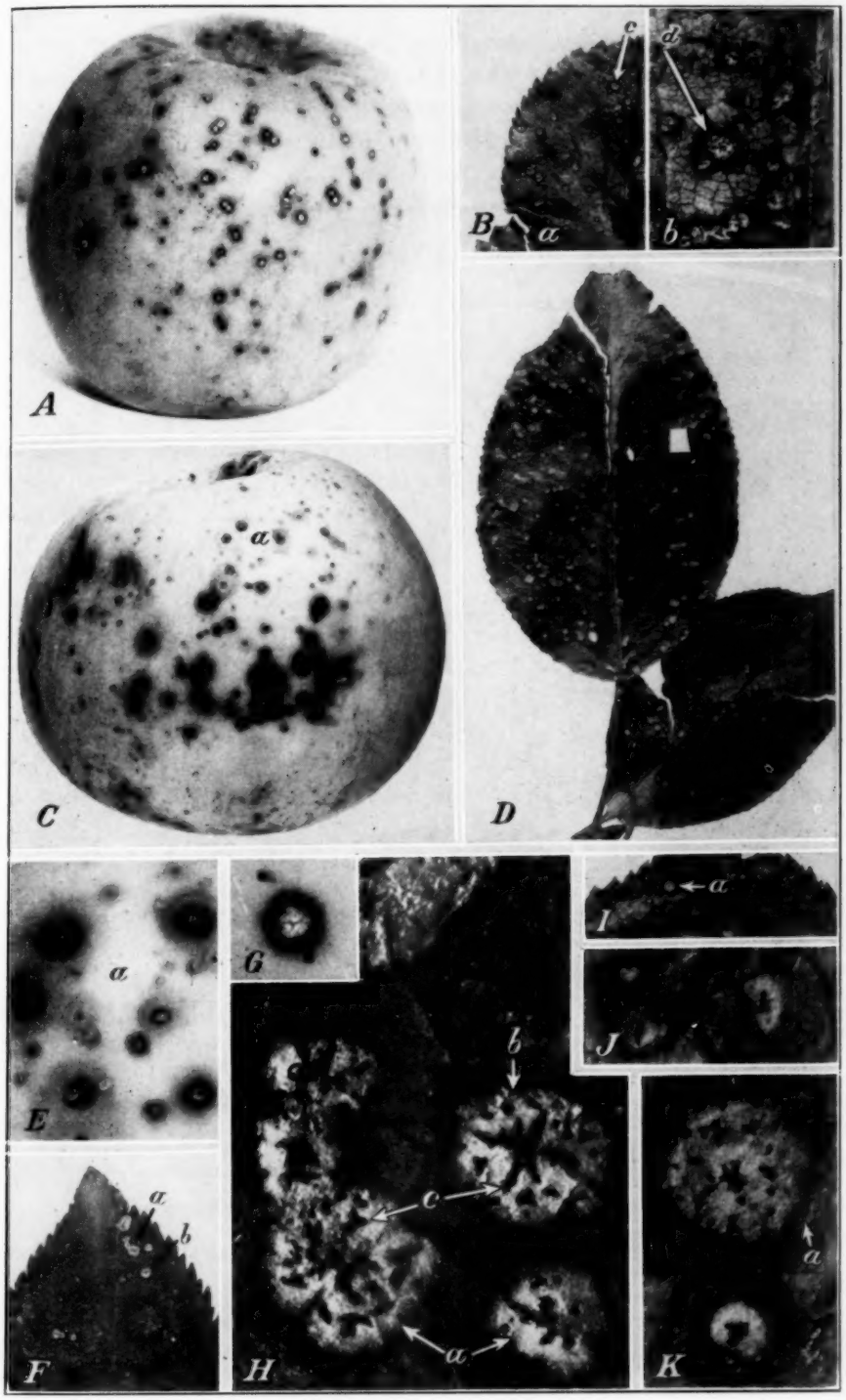
This paper presents certain basic data on the identity and known North American distribution of *Elsinoë piri* (Woronich.) Jenkins (3, p. 696) previously reported only in abstract form (2, p. 969 and 7). The recent findings in our Northwest of this pathogen, hitherto reported only from Europe (3) and South America (3, p. 694; 5, p. 34–35; 6, p. 154), are of special moment in view of the economic importance of the susceptible fruit crop in the United States.

The first specimens examined were spotted apple leaves from Mt. Vernon, Skagit County, Washington, September 1, 1943, collected by M. J. Forsell and E. I. Smith, and spotted apple fruits from South Bend, Pacific County, Washington, October 10, 1943, collected by R. F. Wilbur, all three of whom participated in the Special Survey in the General Vicinity of Ports of Entry of the U. S. Bureau of Entomology and Plant Quarantine. These initial findings led to a special survey in 1944 in western Washington and Oregon by L. W. Boyle, of the Emergency Plant Disease Survey, U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering, which was supplemented in Western Washington by M. J. Forsell.

Spotting typically produced by this pathogen is represented by the numerous specimens collected during September, October, and November over extensive sections of western Washington and Oregon. Examples of infected apple fruits and apple and pear leaves are shown in figure 1. Spots on the fruit of the Grimes Golden variety (Fig. 1, A) were generally "brick red"[1] (color reading based on dry apple peel) and pale at the center in the case of the older spots. Spotting was particularly colorful on a seedling apple fruit (Fig. 1, C). Smaller spots on this fresh apple were "pomegranate purple," larger more diffuse spots were "Eugenia red," and centers of older spots were purple brown. In contrast, the healthy apple skin in the

[1] Colors in quotation marks are based on Ridgway (10).

FIG. 1. Specimens of *Elsinoë piri* on apple and pear collected during the survey of 1944. A. Grimes Golden apple, Whatcom Co., Washington, autumn, M. J. Forsell. ×1. B. Apple leaf, between Bellingham and Everson, Whatcom Co., Washington, September 21, L. W. Boyle; *a*, ×1; *b*, part of *a*, showing dark conidial stage on the spots; *d*, same spot as *c*. ×3.5. C. Fruit of an apple seedling, vic. Dayton, Yamhill Co., Oregon, Nov. 21, L. W. Boyle and J. A. Milbrath. ×1. D. Pear leaves, Mt. Vernon, Skagit Co., Washington, September 20, L. W. Boyle. ×1. E. Detail of C, *a*, showing enlarged lenticular perforations. ×3.5. F. Apple leaf from Lynden, Whatcom Co., Washington, September 19, M. J. Forsell and A. J. Hansen. ×1. G. Prominent spot on fruit of Ortley apple variety, Bay Center, Pacific Co., Washington, September 28, L. W. Boyle. ×3.5. H. Detail of F, showing abundant dark fructifications of the conidial stage (*c*) H, *a* and H, *b* (inset) same as F, *a*, and F, *b*, respectively. ×12. I. Spots on apple leaf, South Bend, Pacific Co., Washington, September 28, L. W. Boyle. ×1. J and K. Spots from I showing small, although distinct pustules of the conidial stage; K, *a*, same spot as I, *a*. ×12. Photographs by M. J. Forsell (A) and R. L. Taylor (B–K).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region of the spots was "lumier green" and "light lumier green." What may be of particular significance in connection with the survey is the single collection on quince of what is here tentatively identified as *Elsinoë piri*. The spotted leaves concerned were collected at Olga, San Juan Co., Washington, on June 13, by C. Bodie of the U. S. Bureau of Entomology and Plant Quarantine.

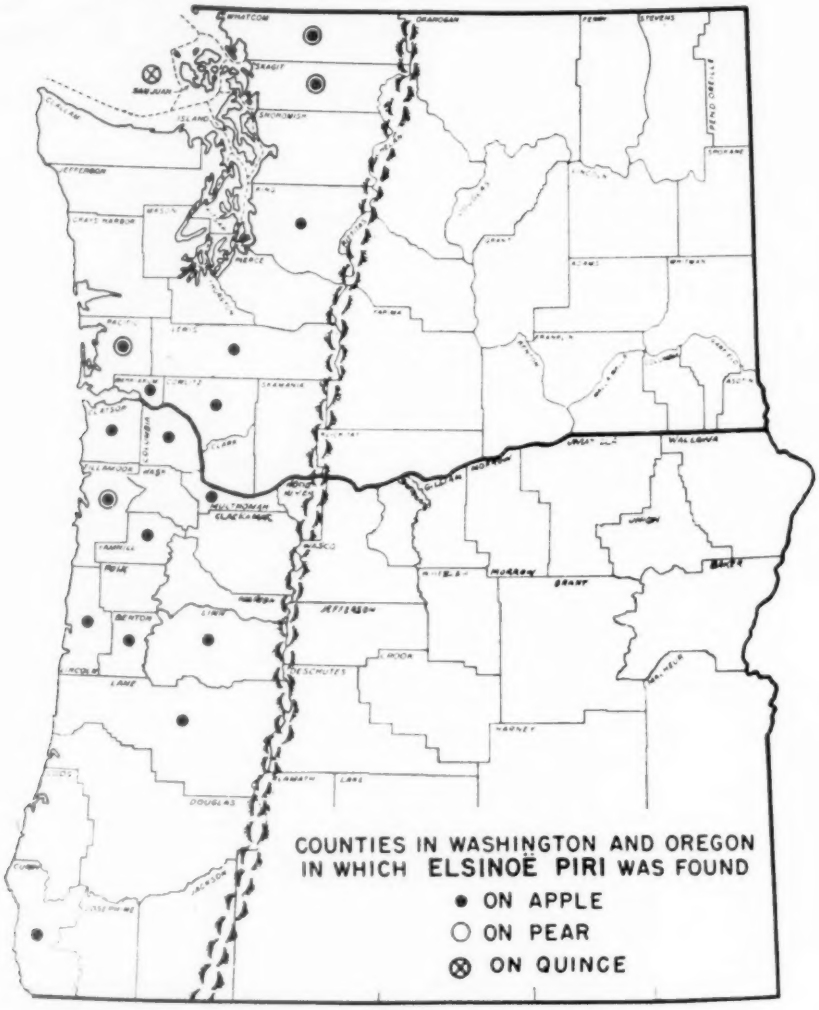

FIG. 2. Distribution of *Elsinoë piri* in Washington and Oregon in 1944.

All the specimens examined showed only the conidial stage, *Sphaceloma pirinum* (Peglion) Jenkins n. com. An isolation was readily made from the fruit illustrated in figure 1, C. On this fruit, vegetative growth of the fungus protruded through the enlarged lenticels (Fig. 1, E), as described by Osterwalder (9) in case of a fruit spot on the Jonathan apple variety.

This spot was subesquently identified by Jenkins and Horsfall (8) as the *Elsinoë* fruit spot. In general on the specimens examined, the conidial stage forms more or less extensive linear, sometimes branched, dark pustules protruding through fissures in the epidermis (Fig. 1, G, also illustrated at a magnification of 12 diameters in 4, Fig. 1, C). Conidia are rarely in evidence. Spots on pear leaves (Fig. 1, D) are similar to those on apple leaves; likewise they agree with Arnaud's (1, v. 2, p. 1059) description of a pear leaf spot in France, which has now been interpreted (4) as that caused by *E. piri.* The specimens and culture here utilized have been deposited in the Mycological Collections of the Bureau of Plant Industry, Beltsville, Maryland. Representative material also is included in fascicle 2 of "Myriangiales selecti exsiccati" by A. E. Jenkins and A. A. Bitancourt, now in process of issue.

The accompanying map (Fig. 2), showing counties in which the fungus was found in 1944, indicates its wide distribution west of the Cascade Mountains. Thus far there have beeen no identifiable findings of it elsewhere in North America.

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OCCURRENCE OF CURLY-TOP VIRUS IN MERISTEMATIC TISSUE

C. F. LACKEY

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INTRODUCTION

The work of Bennett[1] and of Bennett and Esau[2] strongly indicates that the phloem is the tissue in sugar beet and tobacco in which the curly-top virus multiplies. It was demonstrated that the virus reaches its highest concentration in the phloem and that the virus is dependent on that tissue for translocation. Evidence was found that the virus sometimes occurs in the parenchyma but in relatively low concentrations. Anatomical observations indicate that exudate escaping from necrotic phloem may carry the virus into the intercellular spaces of the parenchyma. No explanation is known, however, for the fact that in the crown parenchyma where the cells are closely packed together there was found as much as or more virus than in the parenchyma with its larger intercellular spaces.

This paper reports evidence of virus multiplication in meristematic tissue.

MATERIALS AND METHODS USED IN TESTS ON ROOT TIPS OF SUGAR BEETS AND BEANS

Methods were designed to cut sections of sugar-beet and bean root tips below the protophloem sieve tubes which would contain the meristematic tissue of the growing point. To do this it was necessary to secure data on the distance from the tip of the root cap to the first protophloem sieve tube as well as to the growing point. This information was obtained by sectioning and measuring serial sections of root tips embedded in sets of five.

In order that some correlation could be made between size of root tips and distance to first protophloem sieve tube, the diameter of the tip was measured at the lower end of the first sieve tube. Approximately 500 root tips were examined, including those from both healthy and curly-top infected beets. The diameter of the healthy root tips at the level of the lower end of the first sieve tube varied from 0.31 to 0.66 mm. and the distance from the tip of the root cap to this point varied from 0.55 to 1.20 mm. The diameter of root tips from curly-top infected beets ranged from 0.21 to 0.51 mm. and the distance to distal end of the first sieve tube 0.52 to 1.13 mm.

With these data in mind it was possible to cut sections of root tips well outside the distal end of the first protophloem sieve tube which would contain mainly undifferentiated meristem of the growing point (Fig. 1). The fresh, living root tips were stained lightly with neutral red to make them visible when embedded. They were embedded in warm, soft paraffin in the

[1] Bennett, C. W. Plant-tissue relations of the sugar-beet curly-top virus. Jour. Agr. Res. [U.S.] **48**: 665–701. 1934.

[2] ———————, and K. Esau. Further studies on the relationship of the curly-top virus to plant tissues. Jour. Agr. Res. [U.S.] **53**: 595–620. 1936.

following manner. Commercial parawax was used on account of its pliability at relatively low temperatures. The root tips were placed in a shallow groove in one side of a thin block of parawax. The soft wax was then gently folded to hold them firmly; or, sometimes, two thin blocks of softened paraffin were grooved together over a root tip. When the embedded tips were cooled in the refrigerator for a short time the hardened paraffin held the rootlet firmly without crushing it. By use of a hand microtome and sectioning razor, root-tip sections could be cut from 0.1 mm. up in length. The length of the root tip cut varied according to its diameter, so that an ample safety margin was left between the lowest section cut and the lower end of the first protophloem sieve tube. Large root tips were used. When a section was cut the paraffin was separated from it by means of dissecting needles. From 20 to 132 sections of root tips were used in different tests. These small sections were macerated in a 5 per cent sucrose solution and nonviruliferous leafhoppers were allowed to feed on this solution by using feeding cages of the type described by Bennett.[3] The leafhoppers were then caged singly or in twos on susceptible test beets in the cotyledon stage.

RESULTS WITH SUGAR-BEET ROOT TIPS

By the use of the method described above for cutting these sections of the meristematic region of the diseased beet root tips a series of tests was conducted. As indicated in figure 1, these sections, ranging from 0.2 to 0.4 mm. in length, were from the root-cap portions of the meristematic region and well below the first protophloem sieve tubes. In all 22 of these tests, using from 7 to 24 test beets in each test, positive results were obtained, with an average percentage of infection of 23.3.

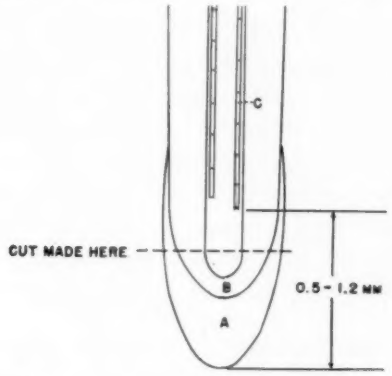

FIG. 1. Diagrammatic sketch of a longitudinal section of a sugar-beet root tip, showing the portion of the tip used in the tests made for the presence of virus. A, Root cap; B, growing point; C, protophloem sieve tube.

Direct Feeding of Vectors on Beet Root Tips

For these tests, large, living root tips from sugar beets infected with curly-top virus were used. These were embedded singly in soft commercial

[3] Bennett, C. W. Studies on properties of the curly-top virus. Jour. Agr. Res. [U.S.] 50: 211–241. 1935.

parawax leaving one-half millimeter or less of the root tip exposed. A short section of small glass tubing was placed over each of these protruding root tips and one or two leafhoppers put in the tube and the upper end closed with cotton. The vectors remained in the tubes 3 to 5 hours at relatively high temperatures. Some of the vectors died and it was difficult to determine the length of time the ones remaining alive had fed on the tips. Some of the leafhoppers remained alive although they did not feed during the 4 hours of the test. Infection was obtained in 3 out of 7 tests made. This again indicates the presence of virus in the meristematic tissue of the root tips.

The possibility that some leafhoppers may have reached a sieve tube when feeding was suggested. To answer this question, studies were made of leafhopper punctures in beet petioles as described by Bennett in 1934. Measurements were made of the depth of these punctures. Many of these punctures were on the side of the petiole away from the vascular bundles and so probably were of the maximum depth possible for the leafhoppers to penetrate. The punctures varied from 0.17 to 0.35 mm. where no phloem tissue was near. The measured distance from the extreme end of protophloem sieve tube to the surface of the root cap varied from 0.52 to 1.13 mm. In the large root tips used in these tests the distance averaged about 1 mm. It seems obvious, then, that the sieve tubes could not have been reached in the feeding process.

TESTS ON ROOT TIPS OF BEANS

Root tips of beans were tested because of their large size and the ease with which they can be grown. They averaged as large as the largest of the beet root tips. These tips were tested in the same manner as the beet tips. The sections of the root tip including the root cap were cut in lengths of 0.4 to 0.5 mm. These were tested for virus content. From 12 to 24 test beets were used in each of 15 tests and infection was obtained in all, with an average percentage of 32.6. As with the beet root tips, these results show that virus occurs in the meristematic tissue of bean root tips.

TESTS OF CAMBIUM FROM TOBACCO STEMS

Types of Plants Used

Two species of tobacco, *Nicotiana tabacum* L. var. Turkish, and *N. glutinosa* L., were used in these tests. When sampled for virus, these plants varied in age, condition of growth, and degree of severity of curly-top symptoms. When inoculated, there was not much difference in the size and age of these plants, which varied from 12 to 18 inches high. However, when cambium was taken from them for sampling, the Turkish tobacco plants were of four classes: (1) Rapidly-growing plants 12 to 15 inches high, 2 to 3 weeks after inoculation, and with severe symptoms; (2) plants 15 to 24 inches high, 3 to 5 weeks after inoculation, and with symptoms past maximum severity; (3) older plants about 24 to 30 inches high, 6 or more weeks after inoculation, showing some recovery; (4) old plants about 36 inches high, 8 or more weeks

after inoculation, and in an advanced stage of recovery with almost no symptoms on the young upper leaves. The *N. glutinosa* plants used in the experiments varied correspondingly in age, size, and length of time after inoculation.

Methods

The bark of a rapidly growing tobacco plant may be readily removed, leaving a layer of cambium tissue on the woody stem. This meristematic

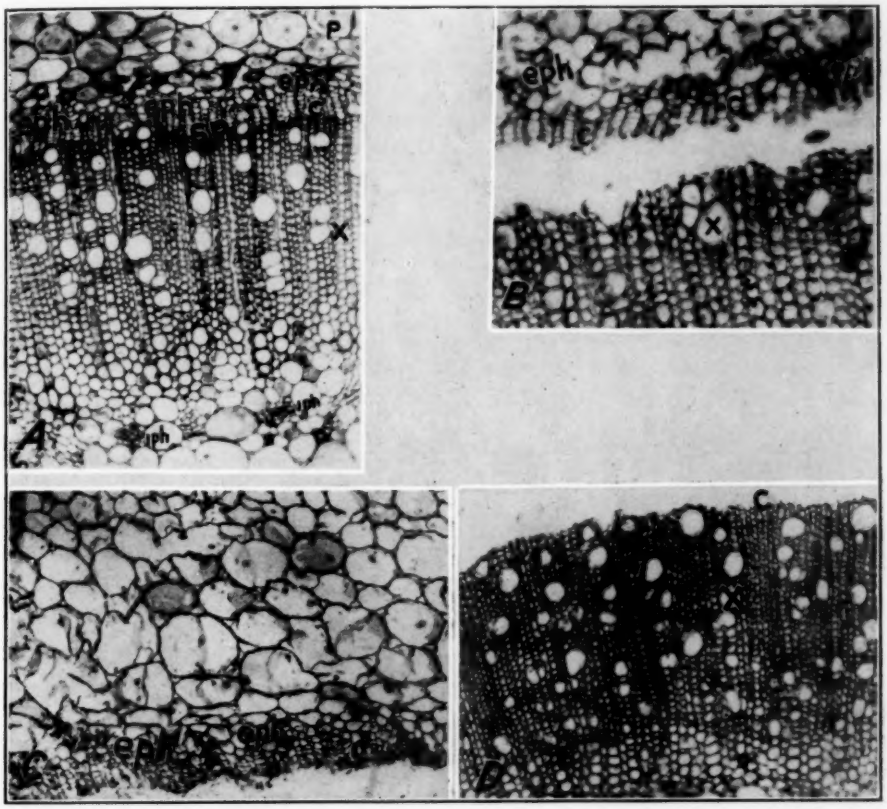

FIG. 2. Transverse sections of tobacco stems: A, Parenchyma cells (p) of bark, external primary phloem (eph), cambium ring (c), point where bark separates from (sp) woody cylinder of xylem (x) and internal primary phloem (iph); B, bark separated from woody cylinder of xylem (x) and showing external primary phloem (eph) in bark at upper edge of cambium layer (c); C, bark of tobacco stem showing external primary phloem (eph) in it; D, woody cylinder showing xylem area (x) and outer edge free from internal primary phloem tissue after bark is peeled off, (c) thin cambium layer.

tissue was scraped from 3- to 4-inch sections of the tobacco stems and the scrapings were suspended in a 5 per cent sucrose solution. Nonviruliferous leafhoppers were allowed to feed on sucrose suspension of the scrapings by using the same type of feeding cage employed with the tests on macerated root tips.

By this method there was little chance of contamination from vascular tissue, as is evident by examination of figure 2. Figure 2, A, is a transverse

section of a tobacco stem showing the bark with external primary phloem, cambium layer, woody xylem, and internal primary phloem. Figure 2, B, illustrates how the bark comes loose from the woody cylinder, leaving only a thin layer of cambium on it and taking all the external primary phloem with the bark. Figure 2, C, shows a section of bark with the external primary phloem and most of the cambium layer pulled off with it. Figure 2, D, is the woody cylinder left after removing the bark with only a thin uneven layer of cambium tissue left on it. Both freehand and prepared sections were examined under the microscope after removing the bark to check possi-

TABLE 1.—*Tests for occurrence of curly-top virus in cambium of Nicotiana tabacum*

Experiment number	Test beets inoculated	Test beets infected	Disease condition of tobacco plants when cambium was tested
	Number	*Per cent*	
1	24	70.8	Young plants 12 to 15 inches high, inoculated for 2 to 3 weeks, and with curly-top symptoms of maximum severity.
2	18	44.4	
3	20	65.0	
4	28	64.2	
5	20	50.0	
6	20	35.0	Plants 18 to 24 inches high, inoculated for 3 to 5 weeks, and with curly-top symptoms a little past maximum degree of severity.
7	18	27.3	
8	20	30.0	
9	20	50.0	
10	24	25.0	Plants 24 to 30 inches high, inoculated for about 6 weeks, less severe symptoms than in the experiments 6 to 9, and first definite signs of recovery evident.
11	20	8.5	
12	20	15.0	
13	20	30.0	
14	20	10.0	Old plants about 36 inches high, inoculated for 8 weeks or more, in an advanced stage of recovery.
15	20	0.0	
16	20	0.0	
17	20	10.0	
18	20	0.0	
19	20	5.0	
20	22	13.6	

bility of some outer phloem tissue remaining on the peeled stem. In every case examined the outer phloem came off with the bark.

Results

The results of these tests with cambium from *Nicotiana tabacum* and *N. glutinosa* are in tables 1 and 2. The concentrations of virus in the cambium were highest in the case of the youngest plants with curly-top symptoms of maximum severity. Apparently the virus concentration in the cambium decreased as the plants recovered.

DISCUSSION

The curly-top virus has certain advantages for the determination of its presence in meristematic tissues. This is due to its inability to infect plants by juice contact, as is the case with some of the mosaic viruses. Since its rapid translocation is limited to the phloem and since there appears to be

little if any of it in parenchyma tissue, there is little chance of contamination in dissecting out the meristematic tissue for testing.

The question arises, why does the virus exist in the meristematic tissue of diseased plants, while there is little or none of it in the parenchyma? In parenchyma derived from invaded meristem the virus must be inactivated or its multiplication stopped.

Relatively high concentrations of virus were found in the cambium near the lower ends of stems of tobacco plants inoculated in the tops. Bennett[4] in his general review of movement of viruses states that present evidence indicates viruses will not move faster than 1 to 2 mm. a day through paren-

TABLE 2.—*Tests for occurrence of virus in cambium of Nicotiana glutinosa*

Experiment number	Test beets inoculated	Test beets infected	Disease condition of tobacco plants when cambium was tested
	Number	*Per cent*	
1	20	50.0	Young plants 12 to 15 inches high, inoculated for about 2 weeks, symptoms at maximum degree of severity.
2	16	50.0	
3	20	60.0	
4	20	70.0	
5	20	65.0	
6	20	60.0	
7	18	33.3	
8	16	62.5	
9	12	66.6	
10	13	30.8	Plants 15 to 20 inches high, inoculated for 3 to 5 weeks, symptoms past maximum degree of severity.
11	20	30.0	
12	20	35.0	
13	20	20.0	
14	20	10.0	Plants 24 to 30 inches high, inoculated for 6 to 8 weeks, in an advanced stage of recovery from curly-top symptoms.
15	20	20.0	
16	20	10.0	
17	20	0.0	
18	20	0.0	
19	20	10.0	
20	20	0.0	

chyma cells. Such a rate of movement would not be fast enough to account for the virus being in the cambium of the lower end of the tobacco stem in such a relatively short time after inoculation even though these plants were only 12 to 18 inches high when inoculated. The probable explanation is that the virus passes from the phloem into the cambium and multiplies in this meristematic tissue. The course of developments in root tips is probably similar.

It seems strange that with fairly high concentration of virus in the cambium and undifferentiated region of the root tips no injury to these meristematic tissues has been observed. Necrosis in the phloem of infected plants is the rule. The few cases of cell degeneration noted by the writer[5] in the

[4] Bennett, C. W. The relation of viruses to plant tissues. Bot. Rev. **6**: 427–473. 1940.

[5] Lackey, C. F. Curly-top virus in root tips of sugar beets and beans. (Abstr.) Phytopath. **28**: 671. 1938.

apparently undifferentiated region of root tips were cells directly below the extreme ends of sieve tubes. The location of these affected cells suggests that they had passed beyond the meristematic stage. Injury to differentiated cells adjacent to the phloem in diseased plants has been commonly observed.

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INJURY TO APRICOT LEAVES FROM FLUORINE DEPOSIT

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(Accepted for publication March 1, 1946)

Injury to dairy pastures and orchards in the vicinity of an aluminum reduction plant was reported in 1943 and 1944. The injury to dairies consisted of the deposit of fluorine on alfalfa and other pasture crops where dairy herds were being kept. This deposit was reported by State authorities as being of sufficient amount to be dangerous to cattle feeding in the exposed fields adjoining the reduction plant and in the direction of the prevailing winds.

Vegetation in the area adjoining the reduction plant suffered to a varying degree. Many of the broad-leaved plants were blackened, and a few English walnut trees and apricot trees within one-half mile of the plant were almost defoliated by the end of July, 1944. Apricot foliage still remaining on the trees was a dull red brown, and the outer edges of the leaves were dead.

Examinations were made at intervals and foliage and fruit sampled from a large orchard property 1¼ miles from the aluminum reduction plant, and directly in line with the prevailing wind. The orchards consisted of 60 acres of White Adriatic figs, 40 acres of Tilton apricots, and 5 acres of Elberta peaches. The part of the fig orchard adjoining the reduction plant was severely defoliated even in August. This varied because the soil is spotted, but there was less defoliation and a better growth of both fruiting wood and the current crop in the half of the orchard furthest removed from the fumes of the plant. The 1943 crop was lighter than in the two previous years, while the growth of fruit wood was shortened and frequently the terminal buds were injured, which resulted in a "witch broom" effect. No discoloration of foliage was noted.

The apricot orchard was very conspicuous from the type of injury occurring. The area adjoining the plant was about 60 per cent defoliated the first part of August. Most of the leaves remaining on the tree were injured and the brown, dead parts of the leaf had an appearance of fire damage. Much gumming was found on the young twigs, and gum pockets were at the crown of some 5-year-old trees. Defoliated areas of 5 to 16 inches were noted on a large proportion of the twigs that made up the bearing wood. Beyond this defoliated area the younger leaves had varying amounts of brown, dead tissue. This type of injury was noted throughout the orchard but was more pronounced adjoining the reduction plant and in spots through the older orchard of 11 to 13 years. Unfavorable soil conditions in gravel spots in the sub-soil or in spots where the soil was too heavy, resulted in an intensification of the symptoms. The 1945 crop was only about 50 per cent of normal; its crop failure apparently being due to the early defoliation and bud injury occurring in 1944. The amount of fluorine present on the leaves was found by washing them in a weak acid solution and determining the fluorine in the

washings by chemical analysis. The range of fluorine found on leaves, taken at random through the fig and apricot orchards, was from 247 to 403 parts per million.

Apricot leaf injury first appeared 2 to 3 weeks after the trees were in leaf. The first symptom was a wilting of the edges, which became progressively more pronounced for about 10 days by which time the tissue was brown and dead. The width of the injury varied from $\frac{1}{8}$ to $\frac{2}{3}$ of an inch, but except in the most severe cases, a varying amount of normal tissue was noted along the central vein. Figure 1 shows the form of injury commonly noted. This type of injury was seen throughout the season. The growing tip of the shoot developed normally and only leaves almost full grown were injured. The damage continued for two years, unchecked, until the plant was shut

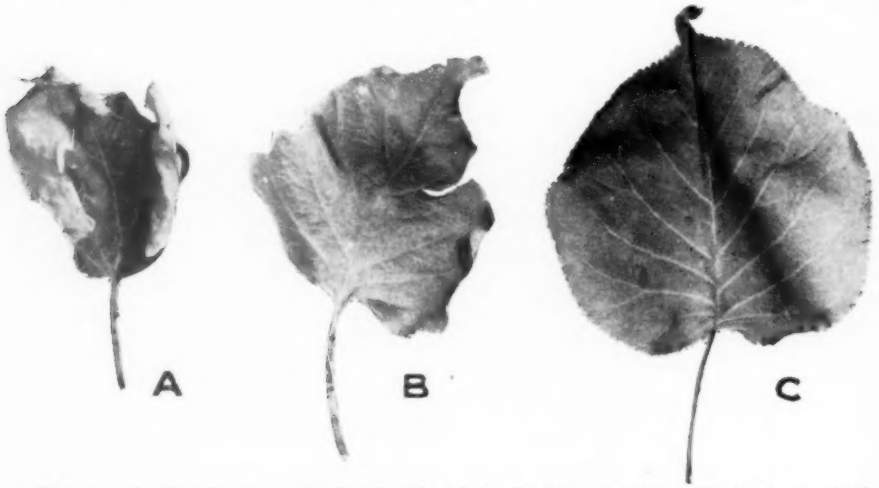

FIG. 1. Apricot leaves. A. Leaf, injured by fluorine deposit, with dead marginal areas attached. B. Injured leaf from which dead areas have fallen off. C. Normal leaf, not injured.

down in August, 1944. Within three weeks following the closing of the plant new growth was normal and no injury to leaves was found afterwards. Similar injury to apricots was noted in a decreasing degree for 8 miles to the windward of the plant. The same type of injury was found on lilac, certain types of blackberry, roses, hydrangea, and a plum tree. No injury was noted on the young peach orchard, although it adjoined the aluminum reduction plant. The injury resulting from the fumes of the reduction plant is distinctly different from boron injury as commonly reported. The first symptom of boron injury is a weakened or dead terminal growth sometimes resulting in dead spikes. Contrasting with this type of injury, the fluorine damage to apricot was on mature or practically full grown leaves. The deposit, probably solid particles with fluorine adsorbed, accumulated on the leaves for two weeks or longer before pathological symptoms appeared. Injury was no doubt intensified by the lack of rain during the dry summer, as in a humid climate the deposit would have been washed off by rain.

Sufficient moisture was present, however, in the dew and fog to dissolve any form of soluble fluorine present.

The presence of fluorine in the fumes emanating from an aluminum reduction plant may result from a disturbance of equilibrium in the electrolytic bath where the bauxite or feldspar ore is treated. A process commonly used in recovering alumina is to fill the pots with a mixture of cryolite ($3\,NaF \cdot AlF_3$) and the aluminum ore. The cryolite is kept in a molten condition and the ore added at intervals. A disturbance of the balance between the fused cryolite and the aluminum ore results in freeing a certain amount of fluorine which would be released through the stacks. The control of flue gases, including the released fluorine, is a practical method of avoiding such injury in fields adjoining reduction plants.

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SEASONAL CHANGES IN BIOLOGICAL EQUILIBRIA INVOLVING TWO CHONDRIOSOMAL SYSTEMS IN VARIEGATED HOSTA

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(Accepted for publication March 12, 1946)

Plastids are known to have certain properties of living organisms (5, 7, 9). They arise only from pre-existing plastids or plastid-forming mitochondria, multiply by fission, and have a definite heredity (5, 7, 9). It has been observed that mutated plastids are the cause of certain types of leaf variegation (7, 9, 12). Recently it has been further shown that these abnormal plastids have an effect on the cells that contain them which is in certain respects remarkably virus-like (11). Thus variegation-inducing plastids (hereafter the abbreviation *vi-* will be used) by their presence in the cell lead to characteristic changes in the development and function of the normal plastids, cell maturation, and cell metabolism (11). Furthermore observed gradations in structural complexity and pathogenic action of the *vi*-plastids in a large number of variegations have suggested more than superficial similarities between variegations of plastid origin, and those due to certain types of viruses (2, 11). Demonstration of ribosenucleoprotein in virus-free plastids (2), confirming observations by Menke (8), furnishes a probable chemical basis for the theoretical heredity-controlling plastogene of Imai (7).

In the origination of variegation it seems probable that the initial mutation would in most cases occur in a single plastid or mitochondrium. In any case the establishment of variegation requires that the mutated plastid-forming mitochondria be able to multiply in the presence of the normal or parent plastidome. Field observations of a plastid-induced variegation in *Hosta* revealed that a seasonal masking of symptoms occurred which was in many respects strikingly like seasonal masking of symptoms in certain virus diseases. Because of the previously demonstrated similarities in cellular pathology in plastid-induced variegations, and certain virus diseases (11), this behavior of variegated *Hosta* was of special interest. Furthermore the material seemed favorable for a study of factors which modify the quantitative relationships between the two plastid systems of a variegated plant during leaf development. The present paper reports results of such a study.

MATERIALS AND METHODS

A white variegated clone of *Hosta japonica* Asch. and Graeb. (Fig. 1) was grown out of doors or forced in the greenhouse during winter. Material for cytological examination was fixed by aspiration in freshly prepared neutral formalin solution (10 ml. of a fresh distillate of a mixture of 37 per cent formaldehyde and an excess of $NaHCO_3$ added to 90 ml. of water) and

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examined without sectioning. Some preparations were kept for later observation by replacing the formalin solution with 10 per cent glycerine. While sectioned material was used, much better results could be obtained with the intact leaf. Yasui (12) employed a similar technic with variegated *Hosta*.

MACROSCOPIC SYMPTOMS OF VARIEGATION

Symptoms of variegation ranged from a uniform sectorial pattern to a highly irregular mosaic of variegated and normal-appearing tissues (Fig.

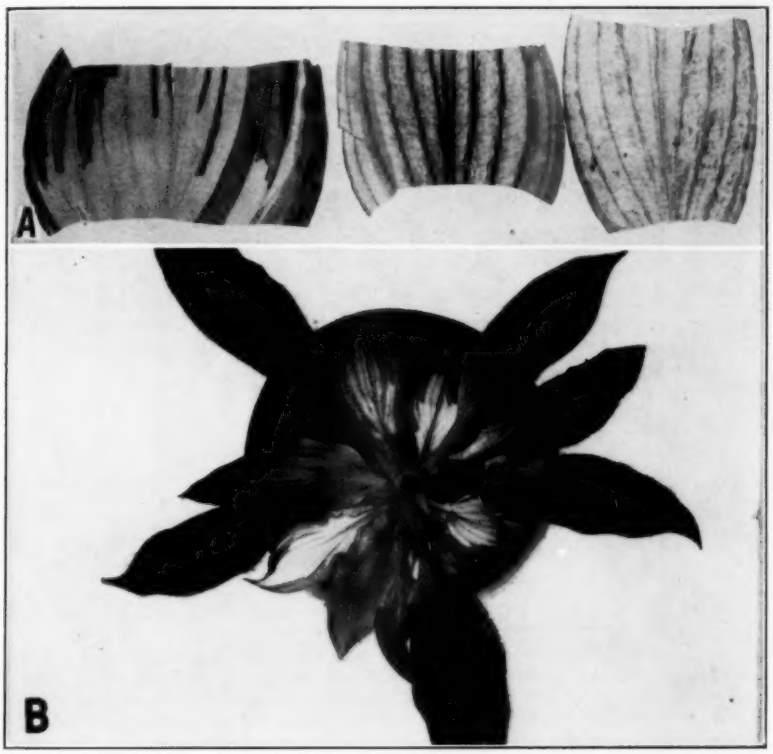

FIG. 1. Variegated *Hosta japonica* Asch. and Graeb. A. Three symptom patterns in leaves formed in spring or early summer. Left to right: sectorial, sectorial-mosaic with green veins, highly irregular mosaic. B. Influence of temperature on symptom pattern. Oldest leaves (green); developed in warm greenhouse, intermediate leaves (variegated strongly); developed in cool greenhouse, youngest leaves (green); developed outside during summer.

1, A). These patterns are due to the distribution of *vi*-plastids which are without visible pigmentation and distinctly smaller than the green plastids (Fig. 2). The contrast between the two types of plastids is always marked, and heterochondric cells (cells containing both types of plastids) are readily distinguished (Figs. 2 and 3). A somewhat similar variegation in *Hosta* has been described by Yasui (12). She noted that the median longitudinal parts of leaves formed in the spring were yellow whereas summer leaves were greenish yellow. Her cytological descriptions, in so far as they cover the

same ground, are in essential agreement with ours. In our clone of variegated *Hosta*, leaves formed early in the growing season were strongly variegated whereas leaves formed during the summer were successively less variegated until some of them appeared macroscopically free of variegation. However, leaves formed in September were often almost as strongly variegated as those formed during the spring. Plants forced from January to March, in a greenhouse held at temperatures approximating those of midsummer, developed leaves with strongly masked variegation (Fig. 1, B). Leaves developed in a cooler greenhouse on the other hand were strongly

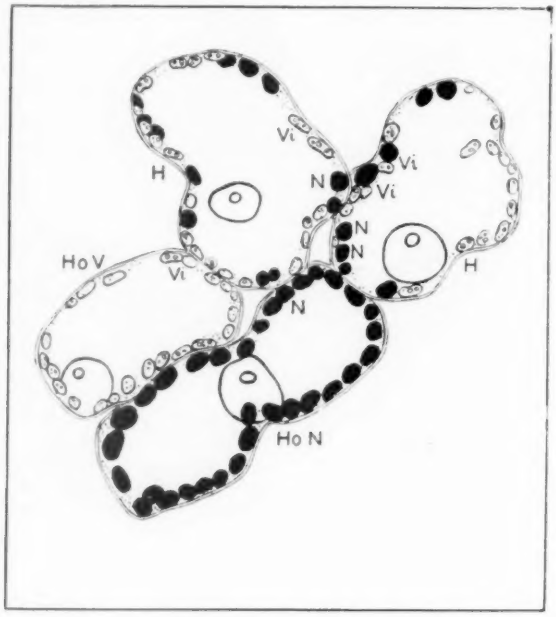

FIG. 2. Four cells from a young summer leaf (July 7) of variegated *Hosta japonica* Asch. and Graeb. These cells occurred in one of the very restricted areas with visible symptoms of variegation. H, heterochondric cell (variegation-inducing plastid labeled *vi*); HoV, homochondric variegated cell; HoN, homochondric normal cell. (Approx. 700 ×.)

variegated. It should be emphasized that throughout the season no change in the symptom pattern of leaves already formed was observed. The masking of variegation apparently took place during leaf ontogeny.

The mesophyll immediately adjacent to the vascular tissues sometimes remained green although the interveinal tissues were nearly or completely white (Fig. 1, A). Microscopical examinations throughout the growing season demonstrated the cytological basis for these variations in symptoms of variegation.

CYTOLOGY OF VARIEGATED TISSUES

Totally variegated areas occurred extensively in spring and early summer leaves. Most cells in such areas contained only variegation-inducing (*vi*-) plastids. Spring leaves with clear-cut sectorial variegation (Fig. 1, A)

were characterized by a preponderance of cells containing only one type of plastid with sharp boundaries between green and white tissues (Fig. 3, C) whereas spring leaves with the irregular mosaic variegation (Fig. 1, A) contained many heterochondric cells (Fig. 3, A and E). In the latter, *vi*-plastids tended to predominate. As the season progressed, successively formed leaves contained variegated areas which were more and more suffused with green. Microscopically this was paralleled by a gradual increase of normal

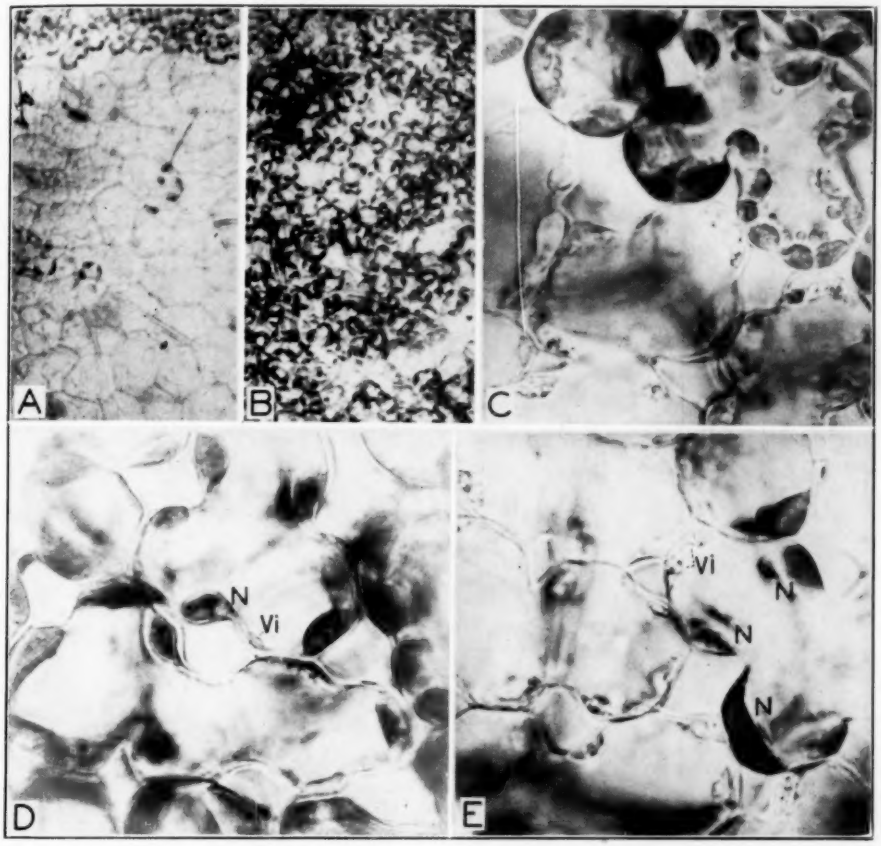

FIG. 3. Photomicrographs of unstained and unsectioned leaves of variegated *Hosta*. A. Heterochondric and homochondric variegated cells adjacent to homochondric normal area in spring leaf. B. Heterochondric area in summer leaf. C. Abrupt transition between homochondric variegated and normal cells of spring leaf. D. Heterochondric cells of summer leaf. E. Heterochondric cells of spring leaf (*vi*-plastids in D and E labeled—*vi*, normal type—N). (Magnifications: A and B, approx. 140; C, D, and E, approx. 900.)

plastids in the abundantly occurring heterochondric cells (Fig. 3, B and D). This decrease in the number of the *vi*-plastids resulted in the formation of large numbers of cells which were free of *vi*-plastids or contained only a few (Fig. 4). This constituted a reversal of the behavior noted earlier in the season. Yasui (12) attributed the deepening of green color in summer

leaves to a growth in size rather than to a change in number or growth rate of normal type plastids in cells with both types of plastids (heterochondric cells). This, however, does not explain the phenomena observed in our material. We did find the plastids of old normal leaves to be much larger

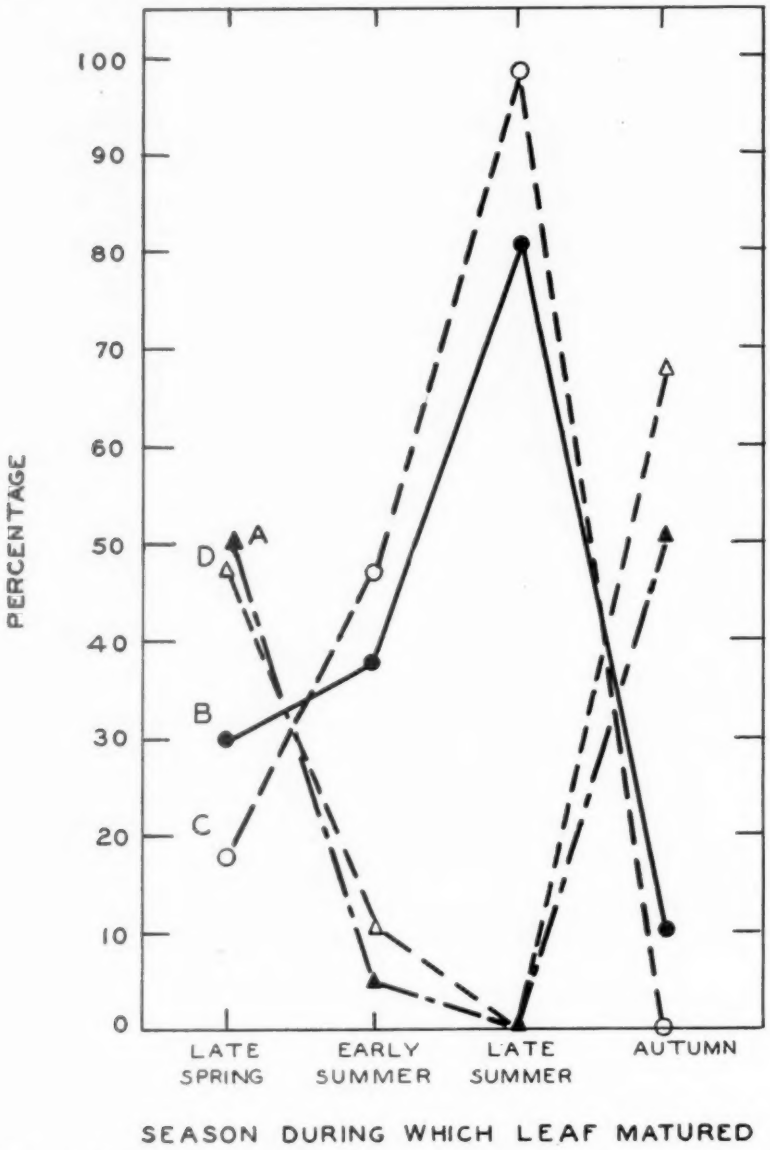

FIG. 4. Influence of season on development of variegation in *Hosta japonica* Asch. and Graeb. A. Approximate percentage of total leaf area variegated. B. Average percentage of a normal green plastid complement in heterochondric cells in measured area (20–25 cells counted). C. Percentage of cells in measured area (of 100 or more cells) that were apparently homochondric normal. D. Percentage of cells in measured area that were homochondric variegated. One typical leaf representative of each season was selected for each set of measurements.

than those of young leaves. Similarly the green plastids of heterochondric cells become abnormally large (2 to 4 times) on aging of the tissues (Fig. 3). These changes in size, however, did not account for the seasonal or high temperature masking, which is due to an increase both in the number of normal type green plastids in the heterochondric cells (Fig. 3, B) and in the number of mature, apparently normal cells (Fig. 4). Some leaves formed during summer appeared macroscopically free of variegation. However, on microscopical examination occasional cells with only one or two *vi*-plastids could always be found in addition to cells in which only normal plastids could be found. In no case was there evidence of plastids of intermediate type. With the reappearance of variegation in September and October many heterochondric cells with large numbers of *vi*-plastids were again observed. Some leaves had large areas of totally variegated cells only.

Cytological examination has shown that there is a tendency for the tissues along the veins occasionally to remain green. Apparently in heterochondric cells close to the vascular tissues conditions are more favorable for the development of normal plastids than for the *vi*-type. Often a rough gradation in the number of *vi*-plastids in the mesophyll cells adjacent to the veins was observed. Thus cells located close to vascular elements appeared either normal or had very few *vi*-plastids, whereas cells further removed from the veins had progressively more *vi*-plastids until homochondric variegated cells occurred mid-way between adjacent veins. The small secondary veins sometimes exerted an influence of a similar nature on the mesophyll (Fig. 1, A). Spring leaves with clear-cut sectorial variegation (Fig. 1, A) were characterized by a preponderance of homochondric cells, both normal and variegated (Fig. 3, A). In leaves of this type there was often little or no observable influence of the veins on cells of the white areas of the leaves. The distribution pattern of the *vi*-plastids, especially the manner in which the veins limit variegated areas, is suggestive of limited intercellular migration of *vi*-plastid forming mitochondria during certain stages of leaf ontogeny as previously noted in other variegations (11). However, proof of such migration must await further work.

DISCUSSION

These studies emphasize the importance of both the external environment (*e.g.*, temperature) and the internal environment (*e.g.*, vascular influences) on the quantitative relationships between the variegation-inducing and normal plastid systems of the cell. The results allow the conclusion that these relationships are in the nature of a biological equilibrium between the cell and the two plastid systems, although the precise nature of this balance remains to be determined. These relationships may be compared with the interactions that exist between a virus and a normal chondriosomal system of an infected cell (10). The interference with the development of the plastids characteristic of certain viruses is well known (11). Since plastids can be considered specialized mitochondria (5) the results might be suggestive

of an approach to a group of animal neoplastic diseases where the existence of similar systems in the cell has been postulated (1, 3, 4, 6).

SUMMARY

White variegated areas of *Hosta japonica* Asch. and Graeb. were due to the presence of colorless plastids which, under certain conditions, multiplied at rates different from the normal plastids in cells which contained both types (heterochondric cells).

In leaves formed in spring or autumn the variegation-inducing plastids apparently suppressed the multiplication of the normal plastids in heterochondric cells and the leaves were strongly variegated. The reverse was true during summer when "masking" of symptoms occurred in successively formed leaves.

The vascular tissues of the leaf exerted an influence on adjacent mesophyll of such a nature that multiplication of normal plastids frequently seemed to occur at the expense of the *vi*-plastids in heterochondric cells regardless of season.

The bearing of these results on cancer and virus problems is discussed.

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PHYTOPATHOLOGICAL NOTES

Flag Smut of Wheat in Mexico.—Flag smut of wheat, caused by *Urocystis tritici* Koern., has never been reported from Mexico prior to 1945. It was first encountered in the region of Zitacuaro, State of Michoacán, on April 27, 1945, and appeared to be distributed throughout a number of fields lying within an area of approximately 10 square miles.

Circumstantial evidence indicates that the causal organism was brought into Mexico on wheat imported from Australia. This wheat was destined for milling, but evidently some found its way into the hands of growers and was used for seed. It is possible that other diseased fields existed during the current year in other parts of the Republic, but no positive evidence was obtained outside of Michoacán.

The Dirección General de Agricultura, Secretaría de Agricultura y Fomento, carried on an eradication campaign in the Zitacuaro area, and a quarantine has been established to prevent the movement of seed wheat from this area to other regions. A campaign of seed treatment has also been begun as an aid in preventing the establishment of flag smut.

The vast majority of the wheats grown in Mexico are susceptible to flag smut. In the event that the eradication campaign is unsuccessful and the disease becomes well established within the country, it will be necessary to begin a program of introduction and breeding for the ultimate selection of resistant varieties.—N. E. Borlaug, J. G. Harrar, and E. C. Stakman, The Rockefeller Foundation, Mexico D. F.

The Effect of Depth of Planting on the Emergence and Survival of Blue Lupine.—One of the most serious troubles in the growing of blue Lupine (*Lupinus angustifolius* L.) as a winter cover crop in Florida is the loss of plants in the seedling stage. After the plants attain a height of 6 to 8 inches, few fail to grow to maturity. Among the organisms isolated from the dead or dying plants, *Rhizoctonia spp.* far outnumber all others. The host-parasite relationship and the factors affecting this relationship are not fully understood. However, it has been observed that fewer plants are lost when conditions favor quick emergence and rapid growth of the Lupine seedlings. One factor affecting the rapidity of emergence and subsequent growth is the depth of planting.

In an attempt to evaluate the effects of the depth of planting on losses from disease, field soil was taken from the surface three inches and put into flats. Sixteen flats were prepared and moved into the greenhouse, where 200 blue Lupine seeds were planted in each flat. In 8 of the flats, the seeds were planted one inch deep while in the remaining 8 flats the seeds were planted two inches deep. Five days after planting 94.1 per cent of the seeds planted one inch deep had emerged; the peak of emergence of 54.7 per cent for the deeper planting was recorded 10 days after planting. A photograph

taken 14 days after planting demonstrates the difference between the two depths of planting in the stand and growth of the seedling plants (Fig. 1).

Some plants were killed by *Rhizoctonia* in all plantings: in the shallow plantings, the seedlings were killed in small localized areas, while only a few scattered plants escaped in the deeper plantings. Twenty-one days after

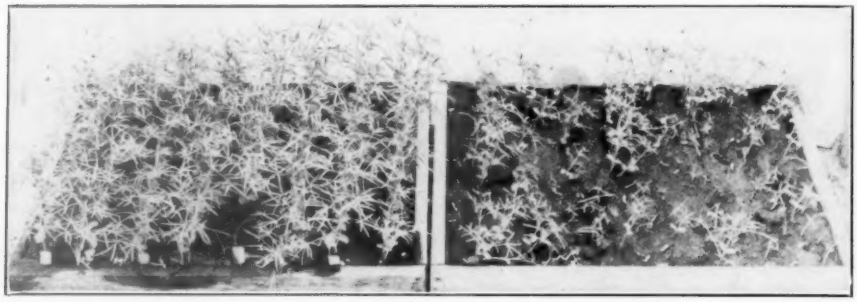

FIG. 1. Blue Lupine plants showing the effect of depth of planting upon stand and growth of seedling plants. Left, seeds planted 1 inch deep. Right, seeds planted 2 inches deep.

planting, when the plants in the shallow plantings had attained an average height of 8 inches, 70.7 per cent of these plants appeared healthy, while only 6.5 per cent of the plants in the deeper plantings were alive.—PHARES DECKER, Florida Agriculture Experiment Station.

A Type of Internal Necrosis of the Potato Tuber Caused by Psyllids.—Manson and Sanford are reported to have found in Canada an internal necrosis of the potato tuber caused by the tomato psyllid.[1] In a recent study of spindling sprout in the White Rose potato[2] an opportunity was afforded to examine for internal symptoms several dozen tubers from plants artificially infested with psyllids. The tubers had been stored at room temperature for about 6 months and either had produced spindling sprouts or failed to sprout at all. On cutting, all these tubers had a characteristic discoloration in the vicinity of the smaller vascular elements. A similar number of control tubers from noninfested plants were free from internal necrosis. The discoloration extended throughout the length of the tuber but was somewhat more prominent at the stem end. On closer examination the discoloration was found to be due to discontinuous dark flecks (Fig. 1, A) rather uniformly distributed in cross section from the main vascular ring to near the center of the tuber. In early stages under magnification an irregular black and white pattern was evident within the flecks. With still higher magnification the cells were seen to contain dark bodies irregular in size, and amorphous to smooth and spherical in appearance. (Fig. 1, B). In later stages, the flecks became more readily discernible macroscopically because

[1] Sanford, G. B., and J. G. Grimble. Observations on phloem necrosis of potato tubers. Canad. Jour. Res. 22: 162–170. 1944.

[2] Snyder, W. C., H. E. Thomas, and S. J. Fairchild. Spindling or hair sprout of potato. Phytopath. 36: (In press). 1946.

of the brownish cast due to cell necrosis, but they lost the characteristic, discrete, dark, intracellular bodies.

It is hoped that the distribution of the flecks and their distinctive microscopic appearance will prove to be of diagnostic value for psyllid yellows where this type of internal necrosis of the tuber occurs.

In other experiments in which spindling sprout was induced by infesting the mother plant with psyllids, internal necrosis was not observed. However, in these instances either the amount of toxin taken up by the tubers

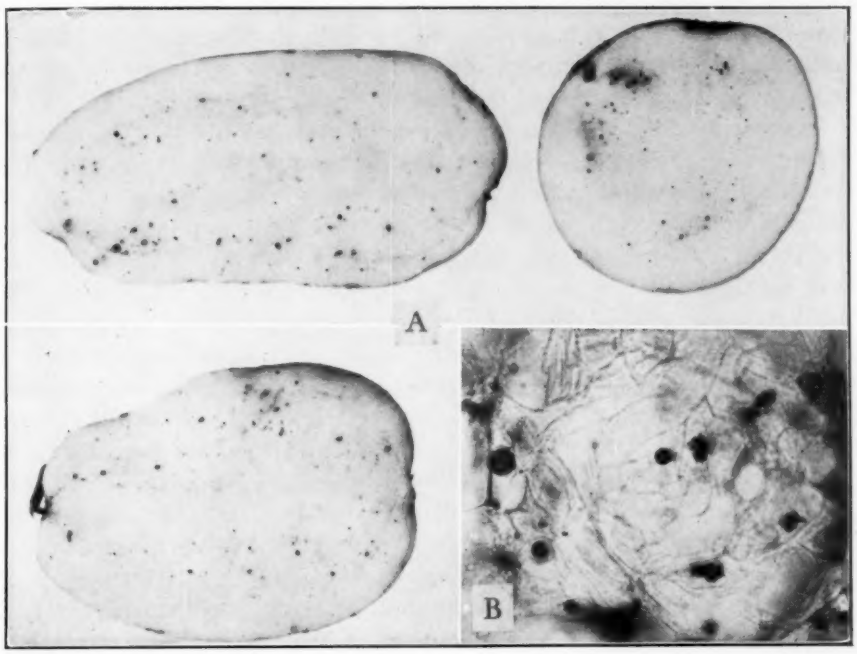

FIG. 1. Internal necrosis of potato tubers from psyllid-infested plants. A, longitudinal and cross sections of affected tubers. B, detail of one of the flecks in A, before complete necrosis, to show several of the dark, intracellular bodies, 180 × (photograph by Catherine Roberts).

may have been less or the tubers may not have been held in storage for a sufficiently long time.

Our evidence does indicate that the kind of internal necrosis illustrated in figure 1 may occur, like spindling sprout, as a symptom of psyllid yellows when tubers produced on heavily infested plants are held in storage.—WILLIAM C. SNYDER, H. EARL THOMAS, and S. J. FAIRCHILD, Division of Plant Pathology, University of California, Berkeley, California, and Mettler Research Laboratory, Santa Maria, California.

Spermogonia versus Pycnidia in Mycosphaerella brassicicola.—A pycnidial stage of *Mycosphaerella brassicicola* (Fr.) Lindau has long been accepted, with the ascigerous stage, as a cause of infection and spread of the

ringspot disease of crucifers. The so-called pycnidial stage occurs abundantly and universally in affected plantings, and has been referred to as a *Phyllosticta*.[1] While making a survey of cruciferous crops for diseases[2, 3] it was observed that ringspot lesions were always evenly spaced and distributed over the affected leaf at random (Fig. 1, A) in a manner expected only where the inoculum is wind-borne. Moreover, no instances were found where secondary lesions were grouped about primary lesions in the type of tear-stain infection usually obtained from water-borne inoculum, such as is the case with the Phoma and Alternaria leaf spots of crucifers. Yet it is the arrangement of great numbers of the so-called pycnidia, and later of perithecia, in each lesion in concentric circles, which is largely responsible for the characteristic symptom and name of the disease.

In the winter of 1945, an examination of numerous infected, living leaves of diseased cabbage plants from the field showed the presence of pycnidium-like bodies and pycnidium-like ooze which corresponded to that previously described for the pycnidial stage.[4] Some of the ooze was collected and mixed with an ascospore suspension prepared from perithecia produced on dead, infected leaves found on the ground beneath diseased plants. This mixed suspension was placed on potato-dextrose, cornmeal, oatmeal, and water agars and incubated both at room temperature, and out-of-doors at Berkeley in winter. Drops of the mixed suspension were also placed on glass slides, with and without bits of healthy cabbage leaves, and on healthy leaves, in moist chambers, under both environments. In all cases germination of the ascospores was obtained within 24 hours (Fig. 1, B) but none of the so-called pycnospores produced any growth in 7 days. Weimer also reported failure to germinate the pycnospores. Furthermore his illustrations of symptoms show only evenly spaced lesions.

Twenty inoculations of detached cabbage leaves were made with ascospores and twenty with the ooze referred to above. Typical ringspot was obtained only where the ascospore inoculum was used, and only where the leaves were incubated out-of-doors.

Twenty single-spore cultures were made from ascospores, and 20 cultures from ooze, on potato-dextrose, cornmeal, and oatmeal agars, and placed out-of-doors. None of the cultures prepared from the ooze grew, whereas all of the single ascospore transfers developed into colonies, and all produced in time the so-called pycnidia, and ooze. Moreover, all the cultures on cornmeal agar produced, in addition, mature perithecia. It is believed this is the first time this fungus has been carried through its complete life-cycle in culture. Since these were single ascospore cultures, *Mycosphaerella brassicicola* is obviously a homothallic fungus.

[1] McAlpine, D. Fungus diseases of cabbage and cauliflower in Victoria, and their treatment. Victoria Dept. Agr. Unnumbered Bul. 38 p. 1901.

[2] Snyder, W. C., and K. F. Baker. Diseases of seed cabbage in California. U. S. Dept. Agr., Pl. Dis. Reptr. 27: 394–398. 1943.

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[4] Weimer, J. L. Ringspot of crucifers caused by *Mycosphaerella brassicicola* (Fr.) Lindau. Jour. Agr. Res. [U.S.] 32: 97–132. 1926.

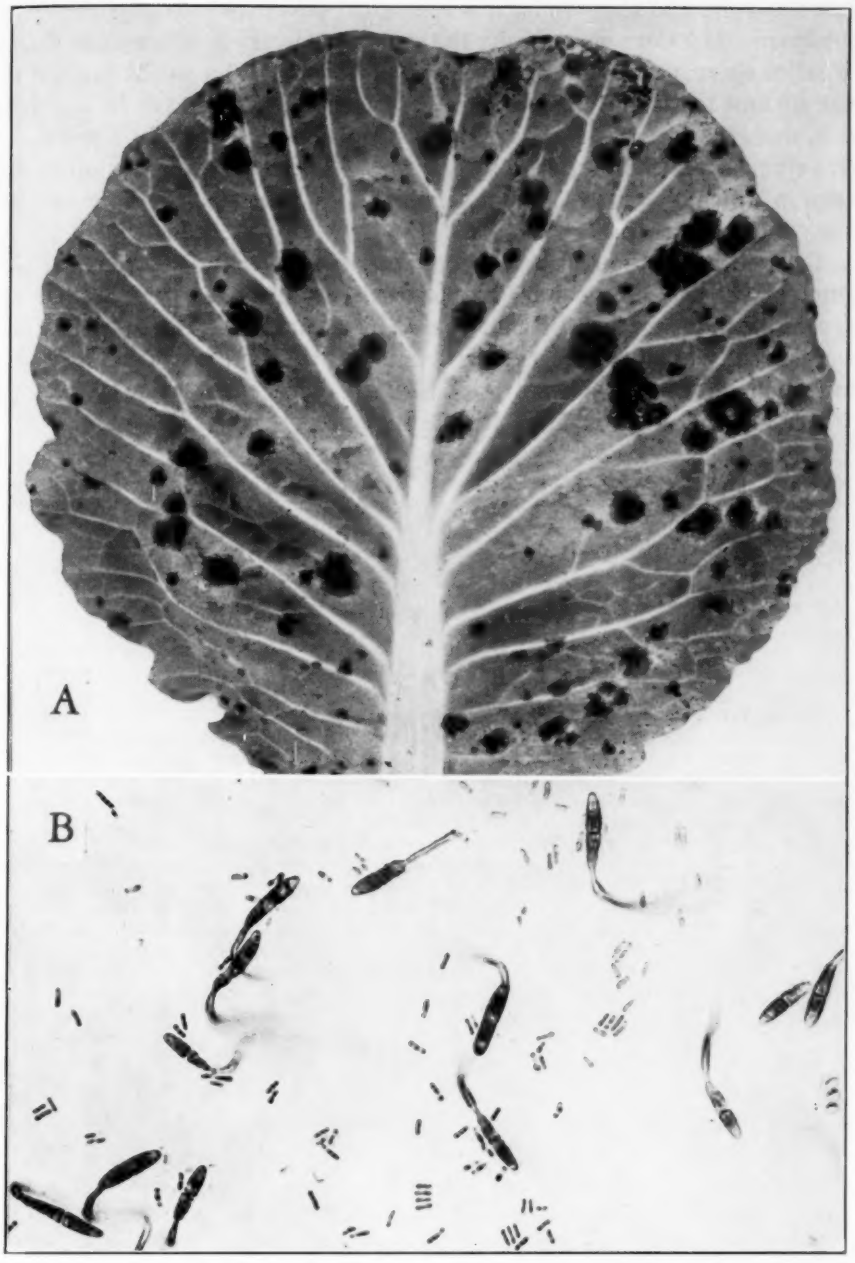

FIG. 1. A. Cabbage leaf naturally infected with ringspot. B. Germinated ascospores and ungerminated spermatia, in water, after 24 hours, 575 ×.

Because of the failure of the so-called pycnospores to germinate or grow in or on various media, or to produce infection on cabbage under conditions favorable to ascospore growth and infection, and because of their character-

istic small size and appearance it is concluded that they are not pycnospores but spermatia. Correspondingly, the structures which produce them should be called spermogonia, not pycnidia. This interpretation would explain the lack of any tear-stain secondary infection on infected leaves in the field. It is, then, the concentric rings of spermogonia on the infected foliage which give ringspot its characteristic appearance. There are probably other diseases in which a spermatial stage similarly has been taken to be a conidial stage of the pathogen.

Recognition of the fact that *Mycosphaerella brassicicola* is a homothallic fungus possessing only one infectious (ascigerous) stage, spermogonia, and no conidia, indicates that dissemination is by means of wind not water. This bears directly upon a correct interpretation of the disease cycle of ringspot, and upon its control.—WILLIAM C. SNYDER, Division of Plant Pathology, University of California, Berkeley, California.

BOOK REVIEW

ANNA WEBER og CHR. STAPEL: *Bekaempelse af Haveplanternes sygdomme. Kortfattede, praktiske Anvisninger.* 11. forøgede og omarbejdede Udgave. Alm. Dansk Gartnerforening. 1944. (Control of diseases of garden crops. Short practical instructions. 11th increased and revised edition, by the Danish Horticultural Association, 1944.)

Everyone who ever has set about to write short practical instructions on the control of plant diseases knows the difficulties to be faced. One cannot expect an average farmer or gardener to know much about phytopathology, therefore one must give as detailed instructions as possible. On the other hand, these must be concise and not too much burdened with theory. The matter must be treated completely, but should not contain materials that are not essential for the practical man. It is not easy to find the right way between theory and practice.

Since this book has reached its 11th edition in 34 years, with more than 70,000 copies, and is known everywhere in Denmark as the "green book," one can presume that the authors have found this right way. And I dare say this little book is really an example of completeness, clearness, conciseness, and the right selection of subjects.

The book is divided into 4 parts. The first deals with diseases and insect pests occurring on many plants (32 pages, 62 alphabetically arranged paragraphs). In the second part the special injuries of the individual horticultural plants are dealt with in alphabetical order of the host plants (115 pages, 417 paragraphs). For each plant a first paragraph gives general instructions on sanitation, then the individual injuries follow in alphabetically arranged paragraphs. The third part contains a survey of the methods of control: cleaning, fertilizing, soil disinfection, seed dressing, dusting, spraying, fumigating, weed control, public control measures, import and export legislation, and compensation (14 pages, 11 paragraphs). In the fourth part (27 pages), finally, there follows an alphabetical survey of the fungicides and insecticides (41 paragraphs), the universal, Danish, English, and American measures and weights, some important addresses, and an alphabetical index.

The attention of phytopathologists all over the world is called not only to this individual book, but to all the practical books edited by workers in the Danish Plant Protection Service. There is no doubt that they are some of the best works dealing with the practical branch of our science. Unfortunately, the Danish language is little known, and this seems to have prevented their more universal use. But this should not be too serious an impediment; everyone who can read English and German will be able to make use of a Danish book, this language being somewhat intermediate between the two last named. And the little difficulty to be faced will be greatly rewarded in this case. Even those who may not read the Danish text will look at these books with great advantage, as they contain a great number of illustrations of an unsurpassed quality.

The following books also deal with the phytopathological and entomological sides of practical plant protection: S. ROSTRUP and M. THOMSEN: Vort Landbrugs Skadedyr (Pests of our agricultural crops), 4th edition, Copenhagen, 1928; M. THOMSEN and P. BOVIEN: Haveplanternes Skadedyr (Pests of horticultural crops), Copenhagen, 1933; E. GRAM and P. BOVIEN: Rodfrugternes Sygdomme og Skadedyr (Diseases and pests of root crops), Copenhagen, 1943; CHR. STAPEL and P. BOVIEN: Mark-Frøafgrødernes Sygdomme og Skadedyr (Diseases and pests of seed-producing crops), Copenhagen, 1943. —H. BREMER, Central Institute of Plant Protection, Ankara, Turkey.